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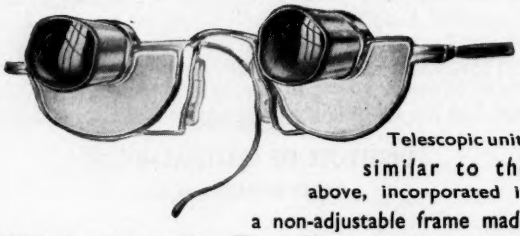

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Brit. J. Ophthal. (1958) **42**, 129.

COMMUNICATIONS

SOME NEURO-OPHTHALMOLOGICAL PROBLEMS*

BY

ALAN MOONEY

Neurosurgical Unit, Richmond Hospital, Dublin

HAVING been associated with the Neurosurgical Unit of the Richmond Hospital for 25 years, I feel that the time is opportune to cast my memory back and recall some of my experiences which may be of interest.

If I succeed in making it clear to the neurosurgeons that the ophthalmologist has a useful contribution to make in the assessment of many of their problems, and to the ophthalmologist that he should be familiar with the neurosurgical implications of certain ocular findings, this paper will have served a useful purpose. To stress the fact that these problems are real—they are a frequent source of anxiety to me—I propose to give just one example of each, although many more are available. The ophthalmologist has an important place in the team, being often the last line of defence the patient must pass before entering the operating theatre. To emphasize this I propose to describe two cases: in the first I was at fault in advising an unnecessary operation and in the second a young girl became incurably blind because an operation was not performed in time.

Case 1, a woman aged 48 years, was complaining of failing vision. There was no toxic history and investigations were negative. This was in 1935 before the days of angiography. Visual acuity was 2/60 in each eye, with slight temporal pallor of each disc.

Perimetry showed bilateral central scotoma embracing the blind spot (Fig. 1). On my advice a right transfrontal exploratory operation was performed. The optic nerves were pale and to the outer side of the right nerve was a hard artery with a bulge on its wall. No tumour was found. The patient died 11 days later.

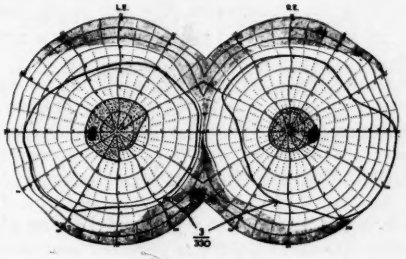

FIG. 1.—Case 1, visual fields.

* Read at the combined meeting of the Society of British Neurological Surgeons and the 36th Annual Meeting of the Irish Ophthalmological Society in Dublin, May 16–18, 1957.

Recommending operation in this case was a serious error of judgment on my part. To-day angiography would be performed on a similar case, and in the event of its being normal, operation would, at least, be deferred.

With all the modern diagnostic aids which are available to-day a negative craniotomy is difficult to justify, but on the other hand it is equally indefensible to allow a patient with papilloedema to become blind without first excluding a space-occupying lesion.

Case 2, a girl aged 17 years, had been seen elsewhere 4 years previously with papilloedema of 2 dioptres and a diagnosis of optic neuritis had been made. When she was referred to the brain unit the visual acuity was less than 6/60 in each eye with left homonymous hemianopia and papilloedema of 4 dioptres with some pallor of each disc. The girl was ataxic with partial deafness in the right ear. A skull *x* ray and electroencephalogram were negative.

Ventriculography disclosed internal hydrocephalus, the third ventricle showing, and at operation a right acoustic neuroma was removed.

The patient is now blind from secondary optic atrophy.

This case brings up the question how long one can allow papilloedema due to a space-occupying lesion to be present without running the risk of blindness from secondary atrophy. Apparently it took 4 years to develop in this patient but I feel that this must be quite exceptional. My impression is that, if papilloedema is steadily increasing, especially when it reaches 4 dioptres, and a space occupying-lesion is suspected, operation should be performed without delay. I have never sought an answer to the time factor because, in order to provide it, one would have to wait until fibrosis had begun and then it is generally too late to save the vision.

Congenital Disc

It is not unusual for a patient to be referred to the Neurosurgical Unit on account of headaches and papilloedema on the assumption that a tumour is present. If the discs are pathological and the patient is not hypertensive, this diagnosis would probably be correct. On the other hand, having regard to the important implications of this finding, the ophthalmologist must be quite sure that the patient *has* papilloedema and not a congenital anomaly.

Case 3, a woman aged 24 years, a postulant in a religious order, was admitted to the Richmond Hospital on account of severe headaches, vomiting, and blindness, with a history of head injury 4 years previously. It was reported that she had papilloedema of 2 dioptres.

When examined by us she said that she could not see light with either eye but the pupils reacted briskly to light. While the discs appeared swollen, the presence of colloid bodies at their margins and the fact that the florid appearance usually associated with papill-oedema was absent, suggested that we were dealing with a congenital anomaly. We also concluded that the blindness was psychogenic in origin. Ventriculography was negative.

A burr hole was made in the right frontal region where the headache was most intense. About 16 ml. subdural fluid was removed and the eyes were bandaged for 48 hours. When the bandages were removed the patient appeared to see normally and the headaches had disappeared. The appearance of the discs did not change.

These "full" discs are by no means rare and are sometimes, but not always, associated with high hypermetropia. The presence of colloid bodies at the disc margin, fibrosis on its surface, and the absence of both a florid, congested appearance and darkening of the veins after they pass over the disc margin should arouse doubts that the condition is pathological.

A certain amount of blurring of the upper disc margin is very common and when associated with headaches should be carefully assessed.

I do not believe that it is possible to give a definite opinion in a large proportion of these cases and my usual procedure is to state that the disc is within normal limits and should be re-examined in a week. If it is then unchanged I repeat the examination 2 weeks later, and if there is no alteration after a further 4 weeks I pass the disc as normal. Further examination is necessary only if the headaches persist after a further 3 months.

Although the vision was not at any time affected, the same point about the discs arose in the following patient:

Case 4, a girl aged 16 years, also a postulant, complained of headaches, and in France, where she was resident, papilloedema was diagnosed. This was subsequently confirmed in England where signs of early secondary optic atrophy were found, and a diagnosis of posterior fossa tumour was made. She was referred to the Richmond Hospital because she wished to return to Ireland.

On examination the discs presented the "full" appearance previously described, and neurological examination and ventriculography were negative.

The headaches cleared up when she returned home, the discs remained unchanged for over 6 months, and she has been well ever since.

Hypertensive Discs

Papilloedema due to hypertension is not uncommon and occasionally patients with this complaint are referred to the Neurosurgical Unit for operation.

Case 5, a patient aged 19 years, was referred to us on account of headaches, vomiting, and dimness of vision. Bilateral papilloedema was noted on admission and the electroencephalogram suggested an intracranial lesion, probably in the mid-line or posterior fossa. Fundus examination showed bilateral papilloedema of 4 dioptres, apparently of long standing, with some pallor of the disc surface. Arterial constriction, venous congestion, and well-marked retinopathy were present.

The patient was obviously hypertensive (the blood pressure was subsequently found to to be 220/150) with albuminuria. I was in some doubt whether the pallor of the disc surface was due to exudation or to early fibrosis, and, while expressing the opinion that the patient had hypertensive retinopathy, I did not completely rule out the possibility of a superimposed space-occupying lesion. This was excluded, however, without resorting to ventriculography, and 8 months later, after medical treatment, the fundi were normal, except for vasoconstriction, and the patient was symptom free. The blood pressure and urine were unchanged.

Similar findings due to a different cause were seen in the following case:

Case 6, a man aged 38 years, had had during the previous 4 months severe bouts of headache, sometimes associated with vomiting and weakness of the legs.

At the time of admission to hospital the blood pressure was 220/140 and there was a

trace of albuminuria. The electroencephalogram showed some diffuse abnormality with no focal features, the appearances being comparable with those found in malignant hypertension.

There was well-marked hypertensive retinopathy and papilloedema of 5 dioptres. The discs were very florid in appearance and, taken by themselves, were more suggestive of intracranial than of vascular hypertension. Ventriculography showed internal hydrocephalus with a patent aqueduct.

At operation a large tumour which proved to be a metastatic carcinoma was removed from the left cerebellar hemisphere (Fig. 2).

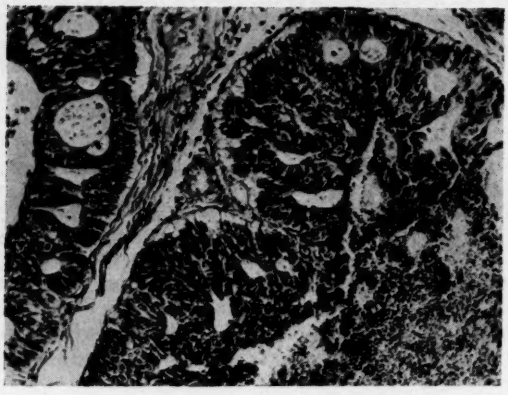

FIG. 2.—Case 6, section of metastatic carcinoma removed from left cerebellar hemisphere.

The foregoing cases underline the problem frequently presented by the "swollen disc". Is it congenital, hypertensive, or due to a space-occupying lesion? I do not propose to repeat observations made by ophthalmologists more experienced than I, but I must say that the more discs of this type I see the more reluctant I am to express an immediate opinion, since diagnostic errors may occur as a result of making only one examination.

When a patient is suffering from headaches, without neurological signs and with blurred discs, there is generally no urgency, and repeated fundus examination will often avoid the necessity for ventriculography or even craniotomy. It is much better that the ophthalmologist should admit that he does not know than that a patient should be submitted to either of these procedures in error through a hasty diagnosis.

PAPILLOEDEMA IN TUBERCULOUS MENINGITIS

We are confronted with a different, and comparatively new problem, when a patient with tuberculous meningitis develops choked discs, since papilloedema from this cause is amenable to neurosurgery.

Case 7, a woman aged 21 years, had been ill for 10 weeks with tuberculous meningitis, and there was papilloedema of 3 dioptres in both eyes with one choroidal tubercle. The oedema increased to 4 dioptres in the course of a week, neurosurgery was advised, and ventriculography showed internal hydrocephalus.

At operation the optic nerves were found to be surrounded by granulation tissue (Fig. 3). This was removed, a third ventriculostomy was performed, and 2 months later the discs were flat and have remained so since then.

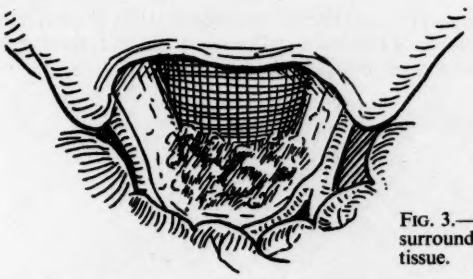

FIG. 3.—Case 7, optic nerve surrounded by granulation tissue.

The next case, which had a less favourable outcome, underlines the danger of assuming, as has been stated from time to time, that papilloedema from meningitis does not develop secondary atrophy.

Case 8, a boy aged 7 years, was admitted to the Richmond Hospital, after having been under treatment for tuberculous meningitis in a County Hospital during the previous 11 months. Fundus examination showed bilateral papilloedema of 4 dioptres in highly hypermetropic eyes. There were signs of beginning secondary optic atrophy and the vision was low. The skull was hydrocephalic and the sutures were wide open.

Operation disclosed fine soft adhesions round the optic nerves. These were removed and a short circuit done on a dilated third ventricle. Just before his discharge from hospital 4 weeks later the discs were flat but well-marked secondary atrophy was present and the visual acuity was practically nil.

There can be very little doubt that a third ventriculostomy, done in time, would have saved this child's sight. The following case shows, however, that conservative treatment is sometimes justified.

Case 9, a woman aged 20 years, suffering from tuberculous meningitis, had double sixth-nerve palsy and 2 dioptres of papilloedema in highly hypermetropic eyes. There were eight choroidal tubercles in the right fundus and five in the left. The discs had not the florid appearance found in the presence of internal hydrocephalus due to other causes but presented a "serous" change such as I have sometimes seen with external hydrocephalus. A chest *x* ray showed miliary tuberculosis.

Daily spinal punctures, together with the usual treatment of Delta-Cortryl, intramuscular streptomycin, P.A.S., and I.N.A.H. were advised.

The papilloedema subsided in 4 weeks, by which time the ocular palsies had also cleared up, and the patient has since made a complete recovery from the meningitis and miliary tuberculosis.

OPTIC NEURITIS

To bridge the gap between the problem of the swollen disc and that of failing vision, the next case showed the not unusual condition of optic neuritis of unknown origin:

Case 10, a woman aged 36 years, gave a history of 5 days' bilateral visual failure and of influenza one month previously.

When she was first seen there was no perception of light in either eye, the pupils were non-reacting, and there was papilloedema of 1½ dioptres with congested retinal veins. The discs did not appear florid. Other investigations, including angiography, were negative.

A course of systemic cortisone and chloramphenicol was given and the swelling subsided in 10 days; 5 months later the visual acuity was counting fingers at 6 metres in each eye and the visual fields showed bilateral central scotoma (Fig. 4).

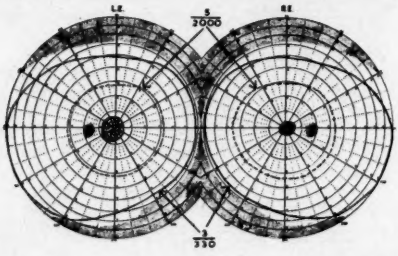

FIG. 4.—Case 10, visual fields.

The vision gradually improved and 1 year and 8 months later it was normal in each eye, the fields being within normal limits, and there was bilateral Grade 2 pallor of the discs with clear margins.

This bilateral condition is not rare in middle-aged patients, and generally a cause is not discovered. Some make an almost complete recovery while others remain blind, both on the same treatment. The rapid visual failure, the appearance of the swollen discs, and the negative angiogram should insure against the risk of these patients being submitted to neurosurgery.

Case 11, a woman aged 48 years, complained of headaches and failing vision in the left eye of 2 months' duration, and 4 months before that loss of the sense of smell and taste during a severe cold. The electroencephalogram was negative, and visual acuity 6/6 in the right eye and counting fingers at 1 foot in the left eye. Papilloedema of 1½ dioptres was present in the right eye, the left disc being normal. The right visual field showed a slight upper temporal quadrantic defect, the left an inferior hemianopia with large macular loss (Fig. 5).

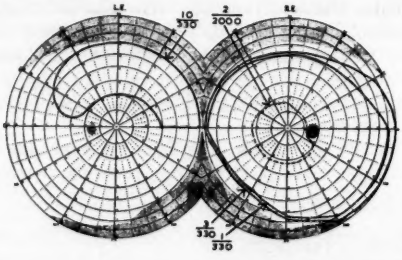

FIG. 5.—Case 11, visual fields.

A left antero-posterior arteriogram showed stretching and displacement of the anterior cerebral artery round a space-occupying lesion in the inferior part of the frontal lobe (Fig. 6, opposite).

A lateral arteriogram showed downward dislocation of the siphon, the anterior cerebral artery being stretched and displaced backwards round the posterior aspect of a space-occupying lesion (Fig. 7, overleaf).

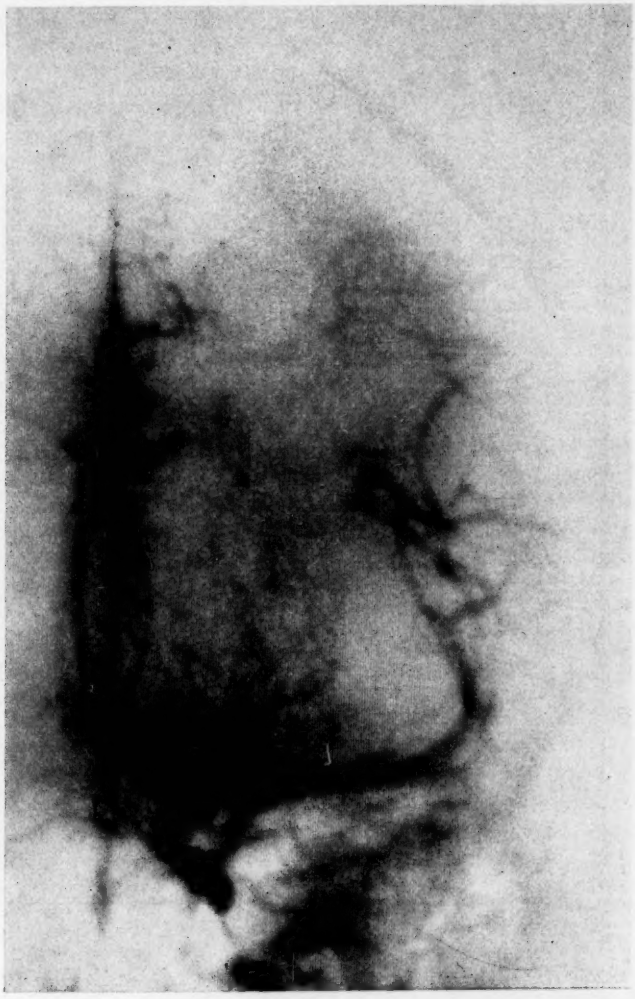

FIG. 6.—Case 11, left antero-posterior arteriogram.

At operation a large left-sided meningioma growing from the cribriform plate was successfully removed.

While the diagnosis of retrobulbar neuritis might have been made in the case of the left eye, the presence of normal vision and the absence of scotomatous changes would hardly justify the application of the term optic neuritis to the right. Positive angiography removed any doubts about the diagnosis in this case just as the negative findings did in the previous one.

I should like to draw attention to the compressed siphon which, in 1948, we put forward as a possible cause of the central scotoma in these cases.

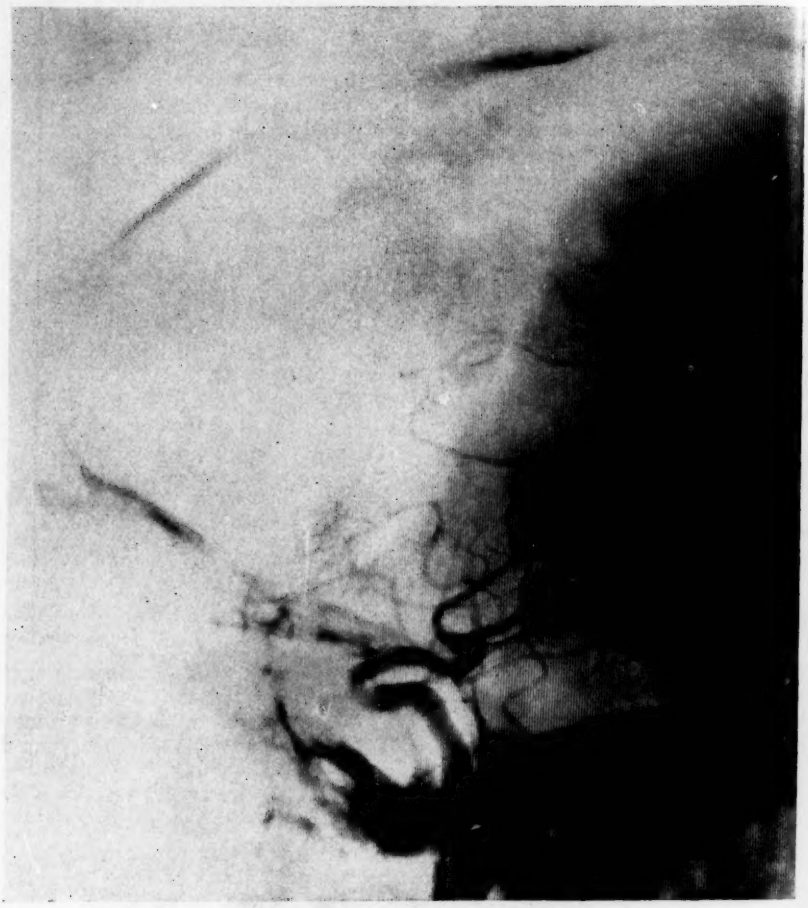

FIG. 7.—Case 11, lateral arteriogram.

REFRACTIVE ERRORS

A high refractive error may obscure the presence of a lesion affecting the lower visual pathway.

Case 12, a man aged 48 years, had complained of failing vision for 2 years, the right eye having become rapidly worse during the past 6 months. He had always been "short-sighted" and had recently consulted a few specialists who had changed his glasses. The visual acuity with correction was perception of light in the right eye and 6/12 in the left. Each eye had a high refractive error, the right more than the left. The right fundus showed Grade 1 pallor of the disc with a shallow complete cup, and the left disc was normal. The intra-ocular pressure was normal.

Perimetry disclosed a left-sided temporal hemianopia, most marked in the upper quadrant (Fig. 8).

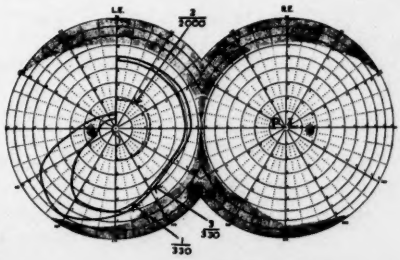

FIG. 8.—Case 12, visual fields.

A right antero-posterior angiogram showed the proximal part of the anterior cerebral artery to be tilted upwards and badly filled (Fig. 9).

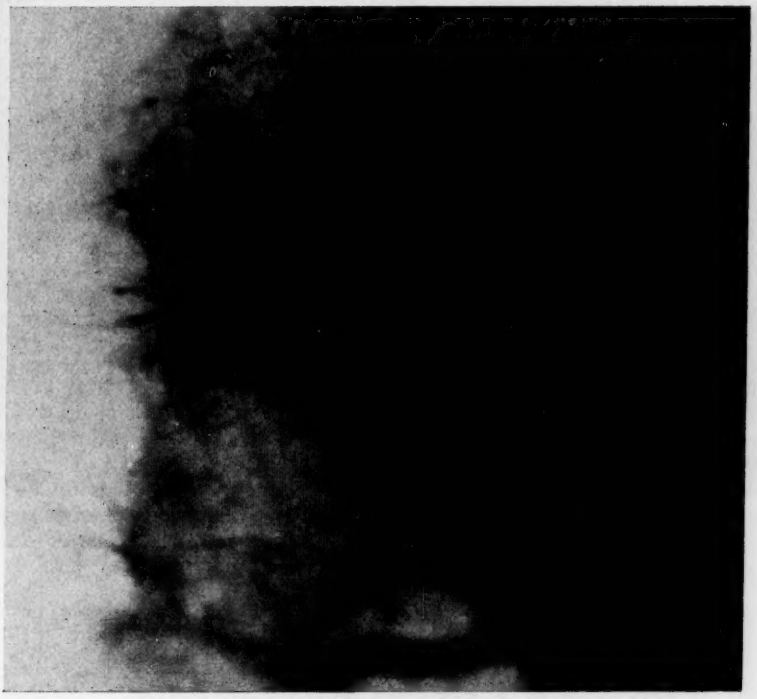

FIG. 9.—Case 12, right antero-posterior angiogram.

A lateral angiogram showed the siphon to be lifted upwards and forwards (Fig. 10, overleaf). At operation a chromophobe adenoma was found beneath the chiasma and right optic nerve, extending lateral to the internal carotid artery, which was embedded in a deep groove (Fig. 11, overleaf). This middle fossa extension was bulky and over its upper pole were stretched the middle and anterior cerebral arteries. The tumour was partially removed.

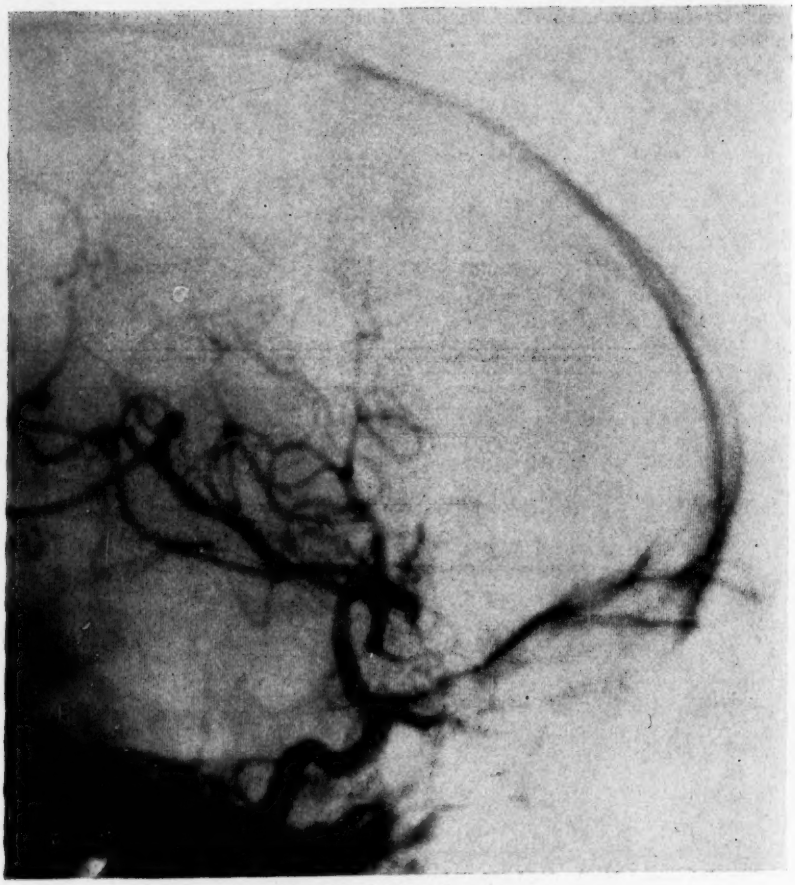

FIG. 10.—Case 12, lateral angiogram.

FIG. 11.—Case 12, chromophobe adenoma beneath the chiasma and right optic nerve.

Before the right eye became blind it would have been very difficult to make a correct diagnosis in this case unless perimetry was performed. It would be reasonable to expect a certain amount of amblyopia in each eye, especially the right, because the refractive error had not been corrected by means of suitable glasses in childhood. Defective vision caused by disuse was seen again in the following case:

Case 13, a female acromegalic, aged 52 years, was admitted complaining of headaches and failing vision. Her right spectacle lens was correct and with it the visual acuity in the right eye was normal. For the left eye she was wearing only a simple lens to correct a complex refractive error, and with a correct lens the visual acuity in the left eye was only 6/24. The discs were normal. The right visual field was normal and the left showed temporal hemianopia to small visual angles (Fig. 12).

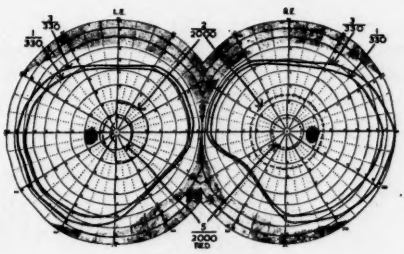

FIG. 12.—Case 13, visual fields.

There was ballooning of the sella with erosion of the floor and dorsum and undercutting of the anterior clinoids. The antero-posterior and lateral arteriograms were normal.

Deep *x*-ray therapy restored the left visual field to normal but failed to improve the defective central vision of this eye which was due to an uncorrected refractive error. The complaint of recent visual failure was due to the onset of presbyopia and not to any affection of the visual pathway.

This case also underlines a common experience with acromegalics, namely, normal discs, minimal field changes, and a normal anterior cerebral artery in the antero-posterior arteriogram.

RETROBULBAR NEURITIS

Chronic retrobulbar neuritis is a diagnosis that should seldom be made.

Case 14, a woman aged 44 years, complained of failing vision in the left eye for 18 months and in the right eye for a few weeks. The visual acuity was 2/60 in the right eye and counting fingers at 2 metres in the left.

The right disc was normal, and the left showed Grade 2 pallor, with clear margins and normal vessels.

The right visual field showed incomplete inferior hemianopia with macular loss, and the left a temporal hemianopia with macular loss (Fig. 13).

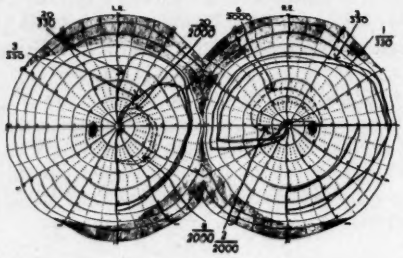

FIG. 13.—Case 14, visual fields.

A skull *x* ray disclosed nothing abnormal.

At operation a meningioma of the tuberculum sellae, the size of a walnut, was found to be displacing the left optic nerve (*Fig. 14*).

FIG. 14.—Case 14, meningioma of tuberculum sellae.

It had originally been suggested that was an early case of disseminated sclerosis, and a similar diagnosis came to *my* mind when I first saw the following patient:

Case 15, a man aged 29 years, gave a history of failing vision in the left eye of *9 days'* duration. The visual acuity was 6/9 in the right eye, and hand movements (in the peripheral field only) in the left. The left pupil reacted *sluggishly to light. The fundi were* normal.

He was admitted to hospital as a case of *acute retrobulbar neuritis and treated* with systemic cortisone, a skull *x* ray disclosed an enlarged sella with destruction of the floor and thinning of the dorsum.

Perimetry, done 6 days later, showed bitemporal hemianopia, the visual acuity being then *6/9 in both eyes (Fig. 15, opposite).*

The left antero-posterior angiogram showed upward displacement of the anterior cerebral artery *(Fig. 16, opposite). The lateral view* showed that the siphon termination was elevated (Fig. 17, overleaf).

Radiotherapy effected a satisfactory visual improvement and 1 year later the visual acuity and fields were normal.

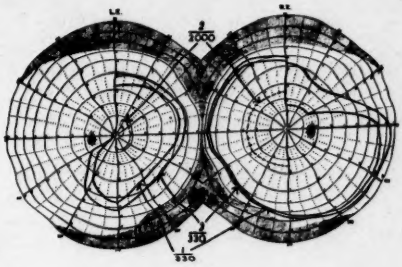

FIG. 15.—Case 15, visual fields.

FIG. 16.—*Case 15, left antero-posterior angiogram.*

FIG. 17.—Case 15, lateral angiogram.

Two lessons may be learnt from this case, the first being that pituitary tumours may simulate retrobulbar neuritis and the second that deep *x* ray is indicated where bitemporal hemianopia is present with normal discs.

There is considerable operative risk in these cases as the tumour is chiefly retrochiasmal and an attempt at even partial removal may disturb the hypothalamus sufficiently to cause death. No such risk is involved in modern radiotherapy and Case 15 shows that the result may be satisfactory if the treatment is given early enough. This case also demonstrates that a central scotoma may be caused by the opening out of the siphon as well as by its compression.

OTHER MISLEADING SIGNS

In Case 16 an unverified subchiasmal tumour simulated a retinal vascular lesion:

Case 16, a woman aged 32 years, stated that one year previously she had noticed that the vision of her left eye was defective. As there was pallor of the left disc, an irregular inferior nasal retinal artery, and a corresponding upper temporal field defect on the same side, a diagnosis of occlusion of the inferior nasal artery of the left retina was made.

Subsequent ophthalmic examination showed the visual acuity to be 6/5 in the right eye and 6/24 in the left. The right disc was just off normal colour, and the left showed Grade 1 pallor. Perimetry showed bitemporal hemianopia affecting chiefly the upper quadrants (Fig. 18).

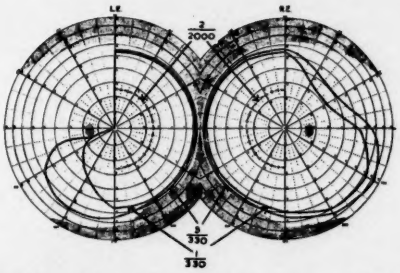

FIG. 18.—Case 16, visual fields.

A skull *x* ray disclosed an enlarged sella with erosion of the dorsum and floor.

A left antero-posterior angiogram showed upward displacement and slight narrowing of the anterior cerebral artery in its proximal part (Fig. 19).

FIG. 19.—Case 16, left antero-posterior angiogram.

The lateral view showed backward displacement of this artery with slight uplift of the siphon termination (Fig. 20).

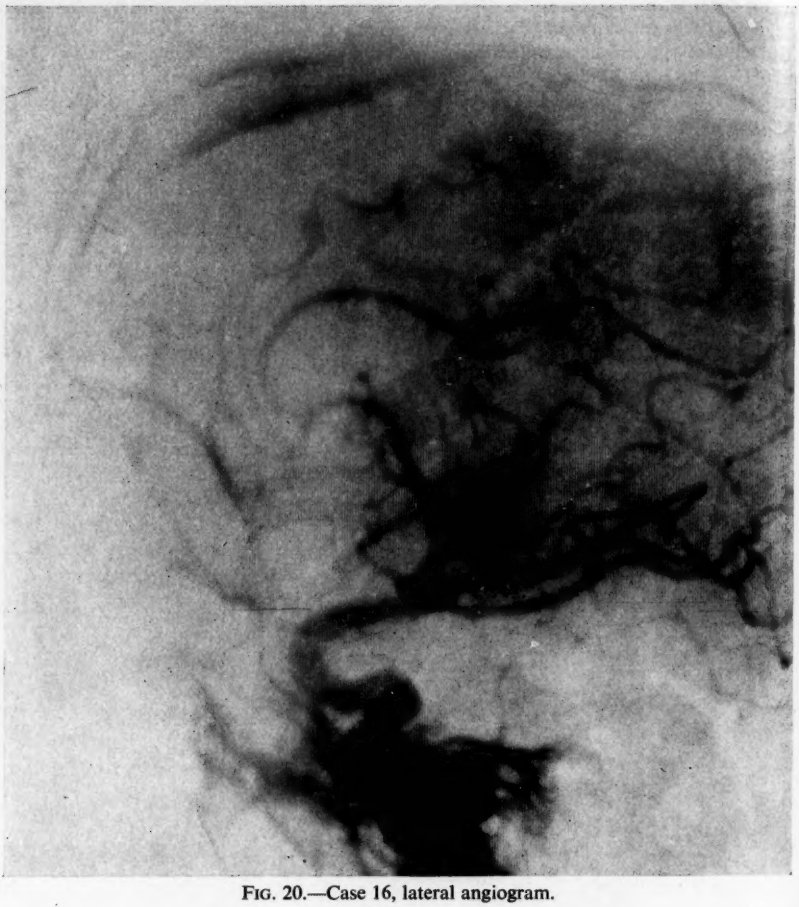

FIG. 20.—Case 16, lateral angiogram.

Both operation and radiotherapy were refused by the patient.

My impression is that where there is bitemporal hemianopia with pallor of one or both discs, surgery is advisable. Even when there is erosion of the sella, meningioma cannot be completely excluded, and if a tumour of this nature is given radiotherapy without improvement and operation is subsequently performed, there is some risk of monocular blindness, as was indeed our experience on one occasion. In the case of adenoma, removal of sufficient tumour to free the optic nerves and no more, followed by radiotherapy, appears to be the ideal treatment. A less conservative approach gains little or nothing and increases the risk of post-operative mortality. We have found that pallor of the disc without scotomatous

changes is usually due to lateral displacement of the optic nerve and is associated, in the majority of cases, with the lifting up of a badly-filled proximal part of the anterior cerebral artery.

The symptomatic onset of pituitary tumours takes various forms and led in Case 17 to a diagnosis of Adie's syndrome.

Case 17, a woman aged 29 years, gave a history of diplopia and severe right-sided headache of 18 months' duration. The right pupil had become enlarged and the vision of this eye blurred before our examination. Visual acuity was normal in each eye. There was right-sided semi-mydriasis and accommodation weakness. The discs were normal.

Perimetry showed that left homonymous hemianopia was present for small visual angles (Fig. 21).

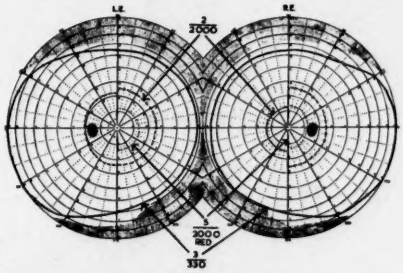

FIG. 21.—Case 17, visual fields.

A skull *x* ray showed gross destruction of the floor and dorsum sellae.

A right antero-posterior angiogram disclosed upward displacement of a badly-filled anterior cerebral artery (Fig. 22, overleaf).

The lateral view showed a widely-open siphon (Fig. 23, overleaf).

Radiotherapy resulted in the visual fields and accommodation returning to normal, but the pupil was unaffected. The pain and diplopia disappeared.

This case demonstrates that there can be a widely-open siphon without scotomatous changes and a badly-filled proximal part of the anterior cerebral artery without disc pallor. Possibly these signs, especially the pallor, only develop when the vascular displacement has been present for some time.

Case 18 presented some signs suggestive of pituitary tumour.

Case 18, a woman aged 66 years, gave a history of intermittent pain in the distribution of the second division of the left trigeminal nerve. When the pain was present the vision of the left eye became blurred. The vision of the *right* eye had been failing gradually for 2 years.

The visual acuity was hand movements in the lower nasal quadrant of the right eye, and 6/9 in the left eye. Grade 2 pallor was present in the right disc, the left disc being normal.

Perimetry showed complete loss of the right field except in the lower nasal quadrant. The left showed slight temporal hemianopia (Fig. 24, overleaf).

The right anterio-posterior angiogram disclosed a large sacculated aneurysm projecting across the mid-line, and displacing the anterior cerebral artery upwards (Fig. 25, overleaf).

The lateral view showed the aneurysm appearing to come off the termination of the internal carotid and beginning of the posterior communicating artery (Fig. 26, overleaf).

The patient refused operation and 1 year later had Grade 3 pallor of the discs. The right eye was blind and she could only count fingers at 2 feet with the left, there being complete temporal hemianopia.

FIG. 22.—Case 17, right antero-posterior angiogram.

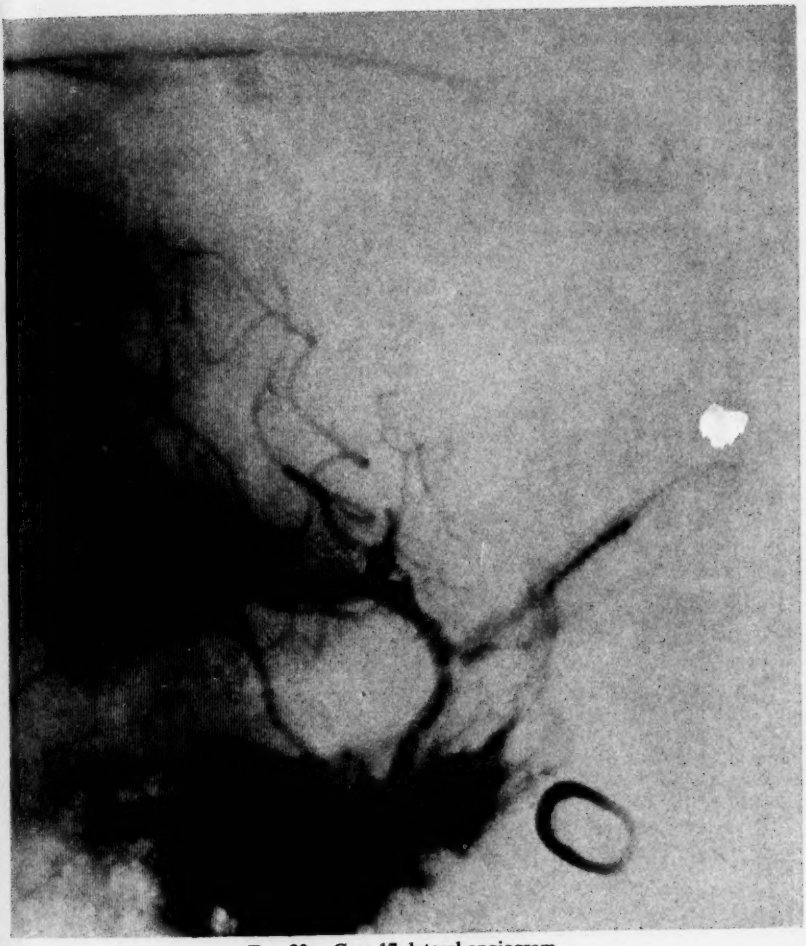

FIG. 23.—Case 17, lateral angiogram.

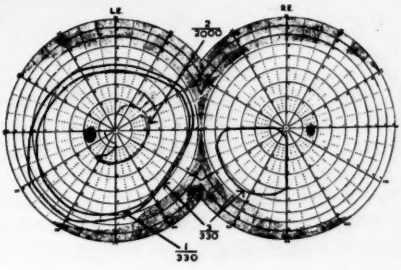

FIG. 24.—Case 18, visual fields.

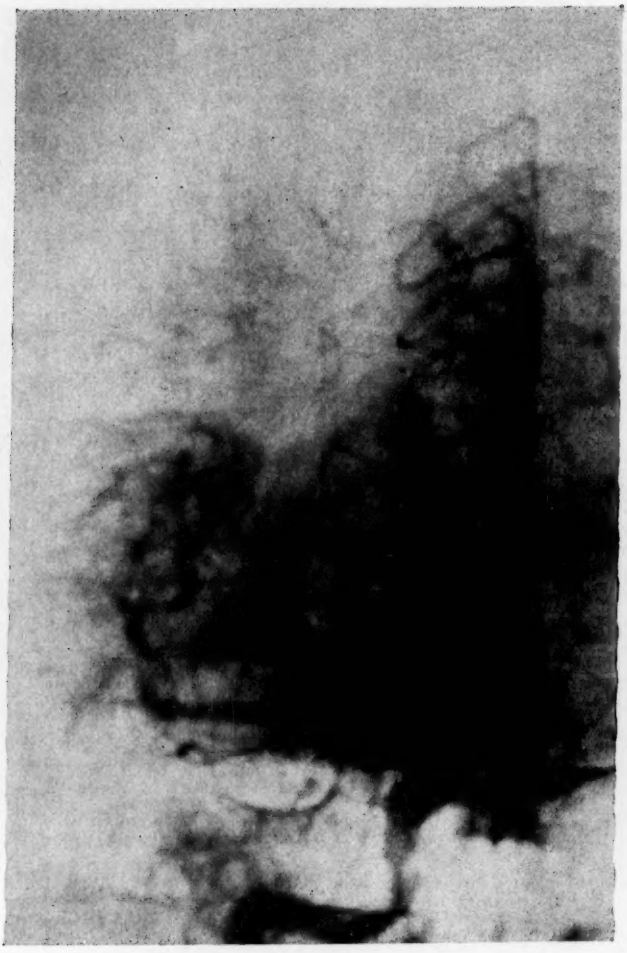

FIG. 25.—Case 18, right antero-posterior angiogram.

At the Congress of the Ophthalmological Society of the United Kingdom in 1956, an ophthalmologist referred to a patient who had been operated on for pituitary tumour when he was in fact suffering from chronic glaucoma. Another speaker, possibly a neurosurgeon, countered this by describing a patient who had been treated for chronic glaucoma and who subsequently died, a pituitary tumour being disclosed at autopsy. The latter experience must surely be most unusual, but the former is quite understandable as is illustrated by Case 19.

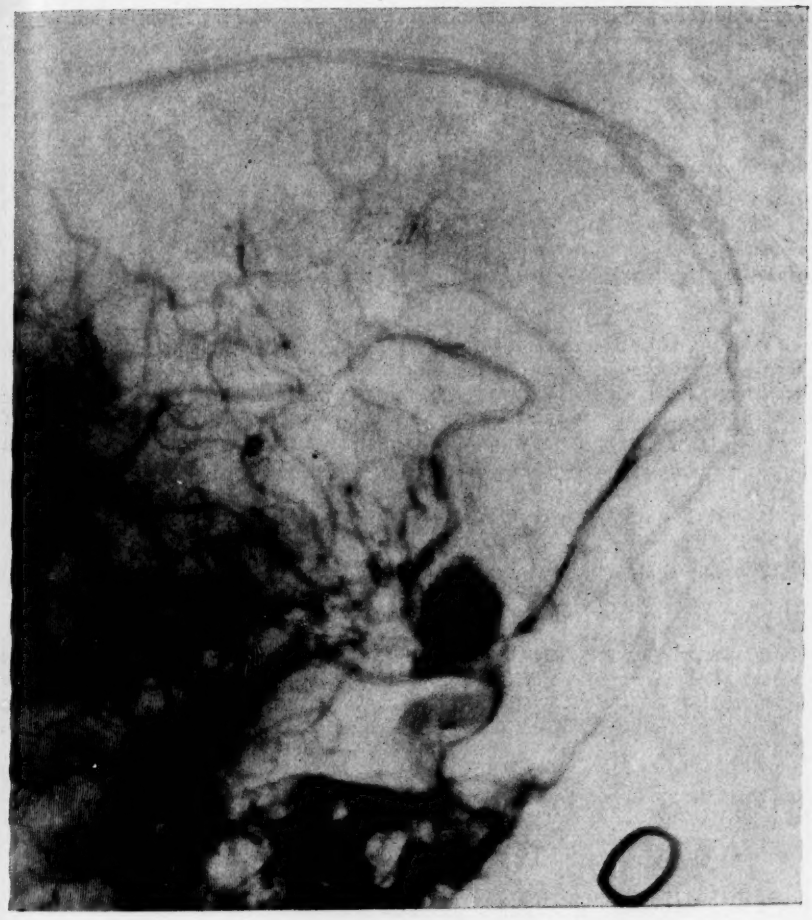

FIG. 26.—Case 18, lateral angiogram.

Case 19, a man aged 43 years, had been blind in the right eye for some years as a result of *chronic uveitis and secondary glaucoma.* The corrected vision of the left eye was normal, but there was marked contraction of the visual field with pallor of the disc. He was referred to the Neurosurgical Unit on account of the possibility of intracranial trouble. The visual acuity was nil in the right eye and normal in the left. The right eye was soft with an occluded pupil, and the left showed a few fine keratic precipitates, Grade 2 pallor of the disc with shallow cupping; the ocular tension was 30 mm. Hg, and rose to 40 mm. Hg 1½ hours after the instillation of homatropine. The left visual field showed marked

nasal contraction with general depression (Fig. 27). A chest *x* ray suggested sarcoidosis.

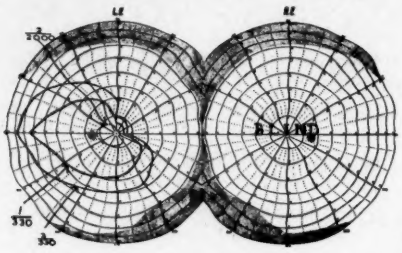

FIG. 27.—Case 19, visual fields.

Cortisone, I.N.A.H., and P.A.S. with topical Pilocarpine and Neo-Cortef improved the uveitis and brought the ocular tension down to 24 mm. Hg.

My impression was that the appearance of the disc and field changes were due to periodic rises in the ocular tension due to uveitis, and as it was not a *straightforward case of* glaucoma I considered that it was a wise precaution to refer the patient to the brain unit for neurological investigation which, however, *turned out to be negative.*

TOXIC AMBLYOPIA

That the gradual visual failure may give rise to diagnostic error is demonstrated *by Case 20.*

Case 20, a man aged 43 years, who had been treated for tobacco amblyopia, gave a history of the left eye having been defective from childhood and failure of the right vision for 3 years. He had consulted two ophthalmologists who had advised him to stop smoking and prescribed tablets. When he was first seen the visual acuity was *nil in the right eye* and 6/60 in the left.

The eyes were hypermetropic, the left being worse than the right. *A slight left internal* concomitant squint was present with defective vision from disuse.

Perimetry showed temporal *hemianopia with macular sparing in* the left eye (Fig. 28).

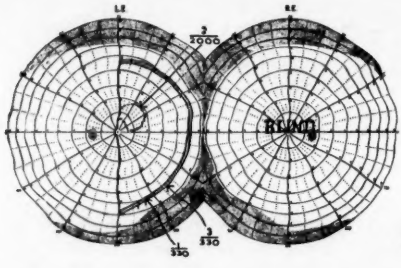

FIG. 28.—Case 20, visual fields.

Grade 2 pallor was present in the right disc, the left being just off normal colour. It was not possible to do a *pre-operative angiography.*

At operation a tumour closely associated with a pulsating swelling was found to be lifting up the right optic nerve *(Fig. 29, opposite).* As it was considered inadvisable to remove the tumour the operation was terminated.

FIG. 29.—*Case 20, tumour closely associated with pulsating swelling found to be lifting up the* right optic nerve.

A right antero-posterior arteriogram showed some lifting up of a badly-filled anterior cerebral artery and lateral displacement of the internal carotid (Fig. 30).

FIG. 30.—Case 20, right antero-posterior arteriogram.

The lateral view showed some elevation of the siphon termination (Fig. 31).

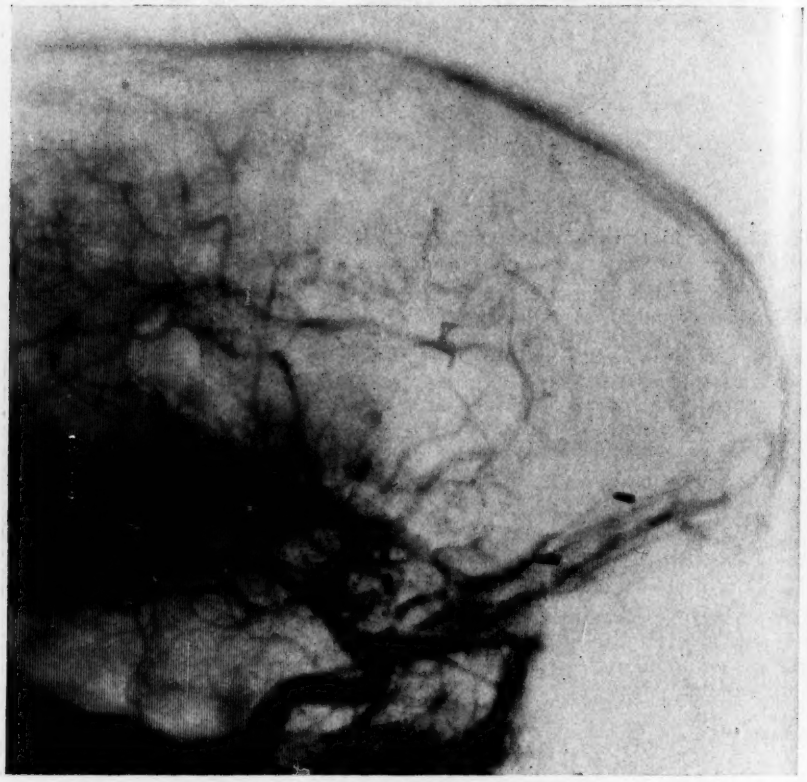

FIG. 31.—Case 20, lateral arteriogram.

After a course of radiotherapy the visual acuity in the left eye was 6/36 with a more suitable correction of the refractive error. The visual field was unchanged.

Case 21 is the counterpart of Case 20:

Case 21, a woman aged 63 years, who complained of failing vision especially for near work during the previous 6 months, was referred to the Neurosurgical Unit as a probable case of pituitary tumour on account of defective vision, bilateral pale discs, and inconclusive fields.

A skull *x* ray showed an extremely thin dorsum sellae, and the visual acuity was 6/36 in the right eye and 2/60 in the left.

There was doubtful pallor of the temporal half of each disc and macular degeneration in the left eye.

The visual fields showed a bilateral centro-caecal scotoma (Fig. 32, opposite).

This lady came from County Kerry and on being asked if she smoked a pipe admitted to smoking 3 oz. of strong tobacco each week. She was advised to stop smoking and sent home. She could not return to Dublin but a letter received from her 6 months later made it clear that she had made a complete visual recovery.

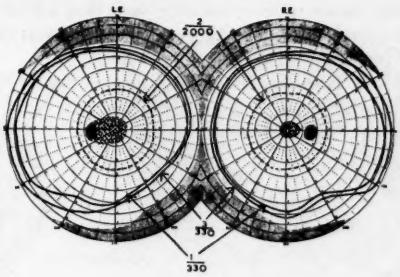

FIG. 32.—Case 21, visual fields.

PALE DISCS IN TUBERCULOUS MENINGITIS

In the early part of this paper I referred to the problem of papilloedema in tuberculous meningitis. The treatment of visual failure in these cases also presents difficulties.

Case 22, a woman aged 29 years, was admitted to Dublin Fever Hospital with headaches of 5 weeks' duration. A diagnosis of tuberculous meningitis was confirmed by guinea-pig inoculation.

She was given intra-thecal and intra-muscular streptomycin, I.N.A.H., P.A.S., and Delta-Cortryl. Within 10 weeks the visual acuity of the right eye deteriorated to hand movements, with subsequent recovery. While this recovery was taking place, the sight of the left eye failed, with simultaneous development of right homonymous hemianopia. Both these defects recovered within 4 weeks. While these visual changes were taking place the discs, from being normal, gradually developed Grade 2 pallor in the right eye and Grade 1 in the left. The patient made an uneventful recovery and the ocular findings before her discharge, 8 months after admission, were normal vision in each eye and Grade 1 pallor in the right disc, the left disc being just off normal colour.

The visual fields showed a slight right upper homonymous quadrantopsia (Fig. 33).

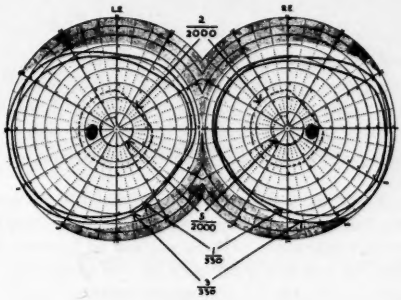

FIG. 33.—Case 22, visual fields. Note that $\frac{5}{2000}$ should read $\frac{5}{2000}$ Red.

The next case presents a contrast with the foregoing:

Case 23, a boy aged 2½ years, was admitted to Dublin Fever Hospital with tuberculous meningitis of 2 weeks' duration, and was given the same treatment as Case 22. The child was blind, with inactive pupils and normal hypermetropic discs. There were no choroidal tubercles. As there was no change in the eye condition after 8 weeks, I advised neuro-surgery and this disclosed an excess of cerebro-spinal fluid in the subdural space.

The optic nerves were covered by very firm adhesions and in places there appeared to be granulation tissue (Fig. 34). A third ventriculostomy was performed after the adhesions had been removed.

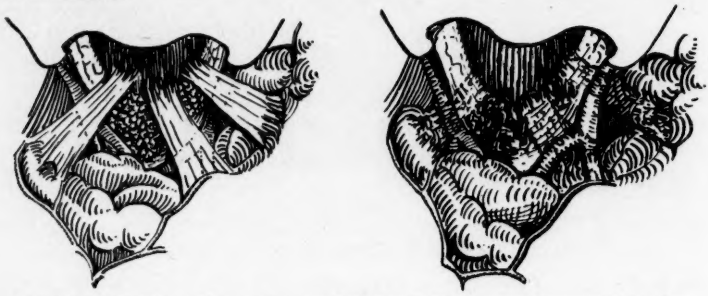

FIG. 34.—Case 23, optic nerves covered by firm adhesions and granulation tissue.

Examination of the granulation tissue showed it to be tubercular (Fig. 35); 3 weeks after the operation the vision began to return and it was apparently normal within a month. There was at no time any apparent abnormality of the discs.

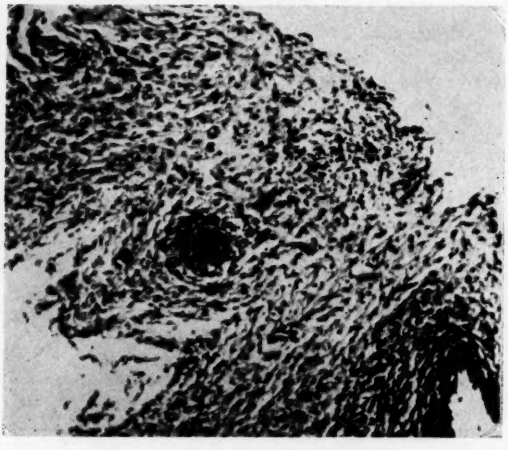

FIG. 35.—Case 23. Section of granulation tissue removed at operation.

Case 24, a boy aged 3 years, was taken into Dublin Fever Hospital with tuberculous meningitis, having been under treatment in a County Hospital for 3 weeks where he was comatose on admission. He was found to be very emaciated, blind, and deaf, and as he was considered too ill for surgery he was given the usual treatment for 3 weeks and was then transferred to the Richmond Hospital.

At operation considerable communicating hydrocephalus was found, with adhesions round the nerves which bled following their removal (Fig. 36, opposite). A third ventriculostomy was performed and the patient returned to the Fever Hospital on the tenth day. With the exception of the Delta-Cortryl, the pre-operative treatment was continued. There was no visual improvement *for over 4 months*, the discs remaining very pale. The vision and hearing then gradually recovered and the boy now appears to see and hear very well and is physically fit. The treatment is being continued.

It may be asked why the first of these three patients recovered normal vision

FIG. 36.—Case 24, appearances at operation.

without neurosurgery in spite of the discs being pale, why the second required neurosurgery to restore vision, the discs being normal in spite of the eyes being blind for 8 weeks, and why the third boy's vision recovered 4 months after operation, the discs being very pale all the time?

These are just three examples of the difficult decisions that have to be made in tuberculous meningitis, not to submit the patient to unnecessary surgery and not to run the risk of irrecoverable blindness by postponing operation for too long.

SUMMARY

If we add up these 24 cases, each to exemplify a different problem, what is the answer? I will try to give it in the following summary.

The ophthalmologist is an important member of any neurosurgical team. His function is to collaborate with the other members in order to prevent an unnecessary craniotomy or to avoid delay when surgery is indicated.

Familiarity with the anatomical variations of the normal disc and discrimination between the hypertensive disc and that due to a space-occupying lesion is essential. It is important to be aware of the danger in allowing secondary optic atrophy to develop even in tuberculous meningitis and also of the type of oedema that will subside without surgical interference.

Correlation of the visual acuity, the fundus appearance, and the perimetric findings should point towards the proper diagnosis, even if angiography is required to finalize it. I am afraid that, in this country anyhow, perimetry must be regarded as a pleasant pastime not covered by the economics of ophthalmology, but it is important, not alone as a means of localizing lesions in the chiasmal region but also, in conjunction with the appearance of the discs, as a guide to treatment in such cases as the following:

(1) Bitemporal hemianopia with normal discs suggests a retro-chiasmal lesion unsuitable for neurosurgery.

(2) Central scotoma in one eye with temporal field defect in the other suggests a space-occupying lesion in the chiasmal region, either elevating or depressing the carotid siphon, usually the former, on the same side as the scotoma.

(3) Bitemporal hemianopia, predominantly non-scotomatous, with some disc pallor suggests that some, at least, of the tumour is pre-chiasmal,

displacing the nerves. Surgery is indicated to verify the nature of the tumour and, if it is an adenoma, to excise just sufficient to free the nerves; it should then have post-operative radiotherapy.

There is no doubt that pituitary tumours may press the optic nerves and chiasma upwards against the anterior cerebral arteries, thereby producing transverse notching of these structures, but the evidence that this notching is responsible for the field defects is not convincing.

Narrowing of the proximal part of the anterior cerebral artery is frequently, but not invariably, associated with the prechiasmal extension of pituitary tumours. This association may have some influence on the development of disc pallor, and possibly also on certain perimetric findings, the anterior cerebral artery being compressed between the optic nerves and chiasma below and the base of the frontal lobe above.

We have seen the anterior cerebral artery recover its normal calibre after the removal of tumour material and this may have some connexion with the recovery in the visual fields and slight improvement that frequently takes place in the colour of the disc post-operatively.

I have not solved the problem of defective vision in cases of tuberculous meningitis; some patients recover good vision after neurosurgery, and others recover without surgery. A third category, usually late cases, fails to improve after operation. Possibly compression of the pial vessels by adhesions determines the loss of visual function, and re-establishment of the blood supply after removal of these adhesions or the arrest of their development following modern therapy may account for the visual recovery.

I found no connexion between therapy and visual failure in these cases.

As patients with tuberculous meningitis are surprisingly good surgical risks, even when pyrexial, I feel that they provide, in the present state of our knowledge, a special exception to the rule that exploratory brain operations should not be advised by the ophthalmologist.

The Future

When B.C.G. inoculation and the rigid isolation of open chest cases have their full impact on tuberculosis, the ocular complications of tuberculous meningitis will disappear from the chiasmal scene.

Other diagnostic problems continue and fresh ones will probably appear. In order to deal with these effectively, more and still more accurate diagnosis, which will both pinpoint the lesion and determine its nature, will be essential.

Success will largely depend on an intimate anatomical knowledge of the visual pathways and their blood supply in relation to field defects and disc changes. When dealing with lesions in the chiasmal region, detailed perimetry, routine air studies, more refined angiography, and a reconciliation of the resulting findings should go far towards accomplishing this end.

My thanks are due to my colleagues in the Neurosurgical Department of the Richmond Hospital, Dublin, for their cooperation, and to Stella Ross for the operation drawings.

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ACUTE VISUAL FAILURE AS A NEUROSURGICAL EMERGENCY*

BY

ALFRED UIHLEIN

Section of Neurological Surgery, Mayo Clinic and Mayo Foundation,† Rochester, Minnesota

MORE often than not visual failure is the result of a disorder of the globe, and hence lies within the province of the ophthalmologist. However, a lesion involving the optic pathways may occasionally be responsible for acute loss of vision (List, Williams, and Balyeat, 1952; Love, Dodge, and Bair, 1955; Uihlein, Balfour, and Donovan, 1957) and at times such a lesion is overlooked because a thorough medical examination was not conducted when the patient first consulted his physician (Baker and Rucker, 1950; Jefferson, 1955; MacCarty, Lillie, Daly, Hollenhorst, and Holman, 1957). Ideally, if irreparable visual loss is to be prevented, the cause should be determined early enough to allow effective treatment. This requires a thorough medical examination, which should include ophthalmological investigation, roentgenograms of the skull, neurological examination, and accurate plotting of the visual fields (Hughes, 1954). In the more difficult diagnostic problems, laminagrams of the skull and cerebral angiography, as well as sagittal sinus venography and pneumo-encephalography, may be required if the condition of the patient will tolerate these procedures (Norlén, 1952).

A review of the records of 310 patients who came to the Mayo Clinic because of unilateral loss of vision was conducted by Uihlein and Weyand (1953). Lesions of structures of the eyeball and glaucoma, in that order, caused impaired vision in 154 (49·7 per cent.) of the patients. Lesions involving the retrobulbar portion of the optic nerve were noted in 108 (34·8 per cent.) of the 310 patients, and additional analysis of these 108 cases showed that in 17·6 per cent. vision was lost because of a pituitary tumour, in 13·9 per cent. because of a meningioma, and in 14·8 per cent. because of an unverified chiasmal lesion. The combined diagnoses of craniopharyngioma, aneurysm, or tumour were made in only thirteen (12·1 per cent.) of the 108 cases. On the basis of these findings it is apparent that the possibility of the presence of a neurosurgical lesion must always be considered in the differential diagnosis of acute visual failure.

On the basis of a review of our patients who have consulted us because of

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† The Mayo Foundation, Rochester, Minnesota, is a part of the Graduate School of the University of Minnesota.

this type of visual failure, we have compiled a Table which we feel represents the most common clinical entities that may produce a sudden decrease in visual acuity. An attempt has been made to list these rare conditions in the order of frequency. Most, but not all, of these conditions will require neurosurgical investigation.

TABLE

COMMON CAUSES OF DECREASED VISION IN ORDER OF FREQUENCY

1. Pituitary tumour
2. Tumour of optic nerve or chiasma
3. Supraclinoid aneurysms
4. Parasellar lesions
5. Carotid arterial thrombosis
6. Third-ventricle hydrocephalus
7. Chiasmal arachnoiditis
8. Fracture of anterior cranial fossa
9. Basofrontal tumour of skull
10. Pseudo-tumour cerebri

It is not my intention to review the diagnostic features in all these conditions, but salient clinical findings in some of them are outlined. The case reports illustrate the diagnostic points that indicated the need for early neurosurgical intervention.

Case Reports

Case 1, a lawyer aged 64, had apparently been well until 8 days before he was admitted to a Rochester hospital. Apart from an incompletely treated syphilitic infection contracted in his early twenties, he had apparently always been in good health.

Six days before admission he began to vomit, 2 days later he noted loss of visual acuity in the left eye, and the next day his left eyelid began to droop. Lumbar puncture and examination of the cerebrospinal fluid (performed at home) revealed increased pressure, with 80 to 90 erythrocytes per c. mm.

On admission he said he was blind in the left eye and that he noted "spotty" blindness of the right eye. He complained chiefly of generalized malaise and headache, with nausea. He had noted a decrease in his sense of smell for about a year.

The patient was tall, with acromegaloid facial features, but the hands and feet were normal. He was drowsy but oriented. Blood pressure 132/80. Pulse rate 68. Except for a bilateral Babinski response, apathy, and Grade 3 palsy (on the basis of 1 to 4) of the third cranial nerve on the left, the results of the neurologic examination were negative. The left pupil was dilated and fixed, and the lid drooped slightly, but perception of light was present in both eyes.

At ophthalmological examination the visual acuity in the left eye was counting fingers and 14/42 (+2·06) in the right. The visual field of the right eye was contracted temporally below. There was no papilloedema.

A roentgenogram of the skull showed enlargement of the sella turcica, with decalcification (Fig. 1, opposite). The pineal calcification was displaced slightly to the left. Results of laboratory procedures, except the results of serologic tests, were normal. The basal metabolic rate was −23.

Two days after he had been admitted to the hospital the patient's condition became worse. Temporal anopsia and a central scotoma on the right developed, and the left eye was blind (Fig. 2, opposite).

FIG. 1.—Lateral roentgenogram of skull in Case 1.

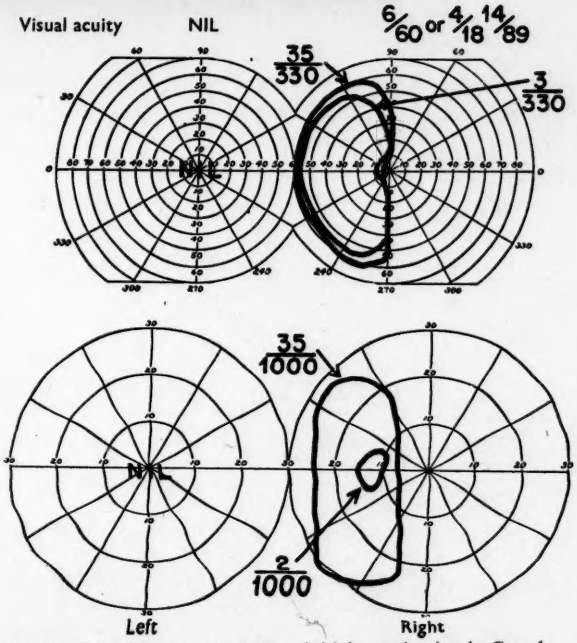


FIG. 2.—Visual fields as plotted on initial examination in Case 1.

Carotid angiograms (*Fig. 3a, b, overleaf*), made with the aid of cross-filling from collateral circulation and with thorium dioxide (thorotrast), excluded an aneurysm of the circle of Willis, but the carotid siphon was deformed as by the pressure of a prechiasmal lesion.

An emergency craniotomy was performed on the left on August 21, 1954. Bloody cerebrospinal fluid was found in the chiasmal cistern. The optic nerves were bowed upward and back by a dark-coloured mass. The left optic nerve was ecchymotic. After

a spinal-puncture needle had been inserted into this mass to exclude the presence of an aneurysm, the capsule of the mass was incised. Much black blood was evacuated in this

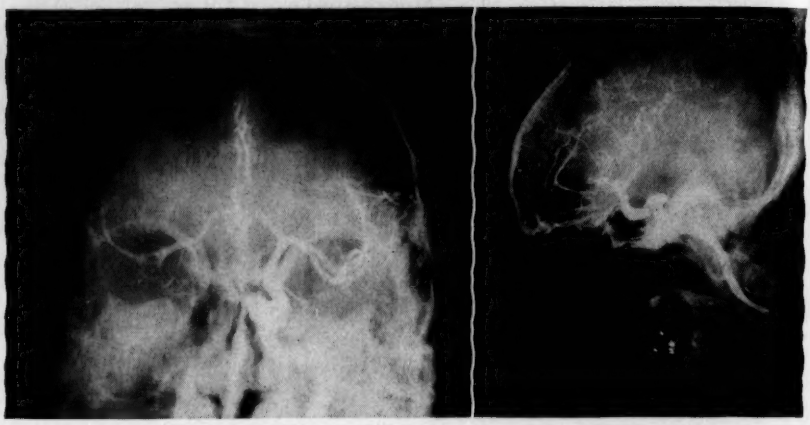


FIG. 3(*a*, *b*).—Antero-posterior and lateral carotid angiograms illustrating elevation of both anterior cerebral arteries in Case 1.

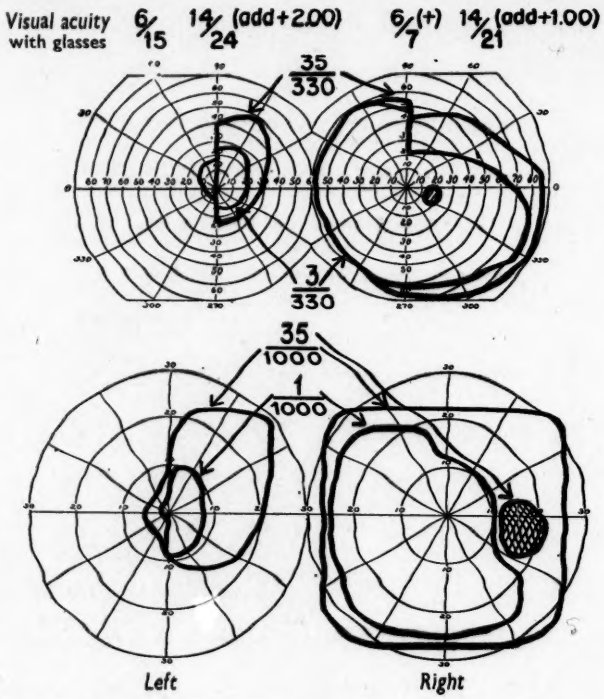


FIG. 4.—Visual fields plotted 9 days after operation in Case 1.

way, and adenomatous tissue of the pituitary gland plus the capsule were removed. All pressure was removed from the optic pathways. The pathologist reported the lesion to be an infarcted haemorrhagic adenoma of the pituitary body.

Convalescence was uneventful. Perception of light on the left was noted by the patient at the end of the second post-operative day, and the third-nerve palsy was lessening. Nine days after the operation marked improvement in central vision in the left eye was evident, with a filling-in of most of the right temporal field and much of the nasal field of the left eye (Fig. 4).

A re-examination on December 6, 1954, disclosed Grade 1 pallor of the left optic disc. The right optic disc was normal, the right visual field was almost normal, and the left was filling out (Fig. 5). Visual acuity was 6/12 in the left eye and 6/10 in the right. Re-examination of visual fields on August 10, 1955, showed no change since the time of the last examination.

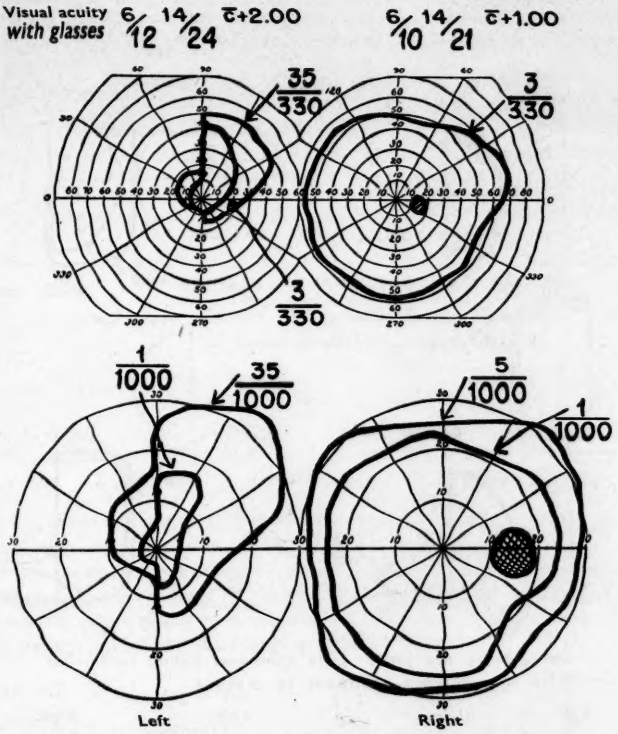


FIG. 5.—Visual fields plotted 4 months after operation in Case 1.

The condition of this patient represents an acute haemorrhage into an unsuspected adenoma of the pituitary body. The vision of this patient was saved by early diagnosis, which permitted definitive surgical treatment for removal of the cause.

Case 2, a boy aged 12, was referred to the clinic on August 24, 1954. The referring physician had diagnosed retrobulbar neuritis; 2 weeks earlier, the patient had noted blurring of vision on the left while target shooting, and 10 days later he could not see objects with the left eye when the right eye was closed, except in the temporal field. On arrival at the clinic, he complained of a throbbing and dull headache, with little or no vision in the left eye.

Results of the physical examination were essentially negative. Roentgenograms of the head and optic canal disclosed no abnormality.

Visual acuity was hand movements at 1 foot with the left eye, and 6/6 and 14/21 without correction in the right. The extra-ocular movements were normal. The ocular tension was 20 mm. Hg each eye. The visual field of the right eye was full, but was confined to the upper quadrants in the left. Both optic nerve heads appeared normal. At this stage retrobulbar neuritis appeared to be the most likely diagnosis.

Two days later the patient complained that vision in the right eye had become blurred; nausea and vomiting began that morning, and lethargy developed. On August 27, 1954, the left eye was blind, and only a residual nasal field remained in the right eye (Fig. 6). The pupils were dilated and equal; the left responded poorly to light.

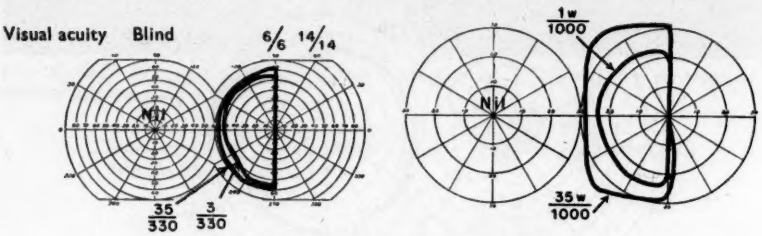


FIG. 6.—Visual fields plotted on August 27, 1954, in Case 2.

An emergency craniotomy performed on the same day disclosed a haemangio-endothelioma of the left optic nerve and left half of the chiasma, with intramedullary rupture of the optic nerve. Acute haemorrhage had occurred within the haemangio-endothelioma (Fig. 7), compressing the anterior portion of the third ventricle. The segment of involved nerve was resected from the globe up to and including that portion of the chiasma on the left. Post-operative *x*-ray therapy was administered.

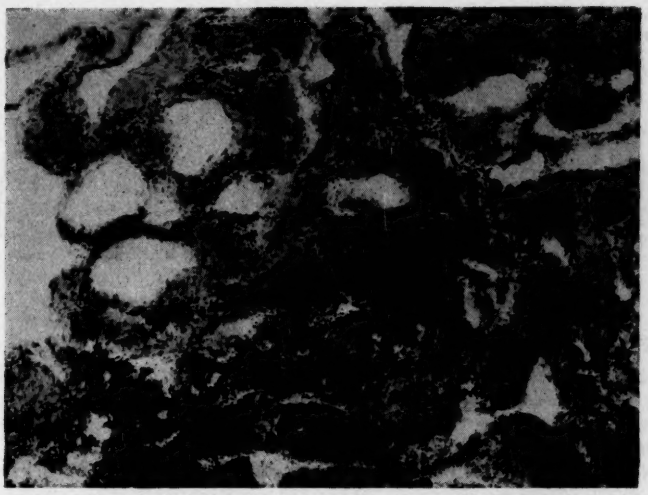


FIG. 7.—Section of pathological specimen in Case 2. Haematoxylin and eosin ×75.

Twelve days after the operation the right visual field had recovered the inferior temporal isle (Fig. 8).

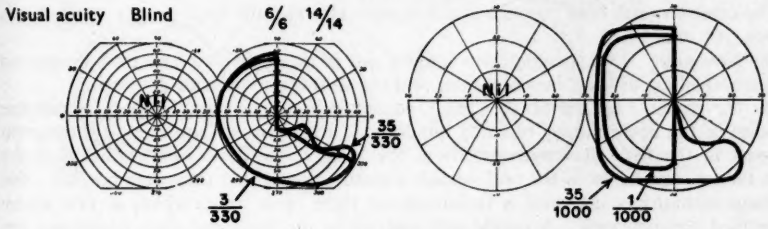


FIG. 8.—Lower visual fields plotted on September 8, 1954, after operation in Case 2.

Four months later there was Grade 1 pallor of the right optic disc, and Grade 3 pallor of the left optic disc (on the basis of 1 to 4). There was no additional loss of vision in the right eye, and the return of the temporal field was of the type that may follow resection of a portion of the left half of the chiasma (Fig. 9*a*).

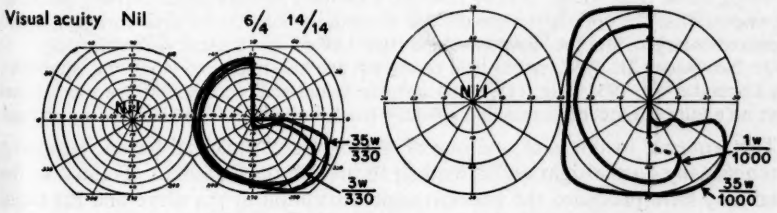


FIG. 9(*a*).—Visual fields plotted 4 months after operation in Case 2.

The visual fields were essentially unchanged when the patient was last examined approximately a year later (Fig. 9*b*).

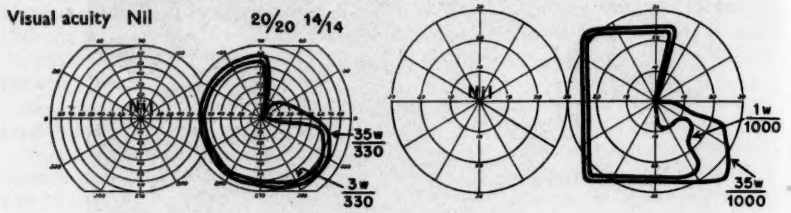


FIG. 9(*b*).—Visual fields plotted approximately 1 year after operation in Case 2 (July 12, 1955).

Case 3, a boy aged 10, was admitted to the hospital on November 6, 1952, with a 5 weeks' history of headaches and progressive loss of vision in the right eye. The boy had been in good health until 5 weeks previously, when his head had been banged against a wall by playmates.

On November 1, 1952, he was suffering from headache and the visual acuity was perception of light in the right eye. The pupillary reflex was down on the right. The visual fields on gross testing suggested upper temporal quadrant anopsia in the left eye. On the next day the anopsia had changed to left temporal hemianopia. The Babinski reflex was elicited on the right. The neck was stiff and the rectal temperature was 102° F. The peripheral leucocyte count was 12,600 per c. mm. blood. These observations were made by a physician in the patient's home town.

The cerebrospinal fluid pressure was 114 mm. Hg, and the total protein content was 55 mg./100 ml.

On November 3, ventriculography (carried out in the patient's home town) suggested inadequate filling of the chiasmal cistern, without shift of the ventricular system.

On November 7 (at the Mayo Clinic), an ophthalmological examination, incomplete because of the acute illness, revealed blindness in the right eye and complete temporal anopsia in the left. Roentgenograms of the head revealed no decalcification of the sella turcica, and views of the optic canals seemed to show that they were normal. Immediate craniotomy disclosed a ballooned-out right optic nerve which at first glance resembled an aneurysm. A needle was inserted in the mass and dark blood was obtained. The nerve had been disrupted by a clot. The operation was terminated at this stage, as the pathologist could not identify tumour tissue in frozen sections. On the seventh post-operative day (November 14), however, the patient became lethargic and experienced Jacksonian seizures of the left arm and left side of the face. Re-exploration at this time revealed bloody cerebrospinal fluid trapped subdurally beneath the frontal lobe. This fluid was released and the optic chiasma was re-explored. The right optic nerve appeared macerated. Biopsy revealed a Grade 1 telangiectatic astrocytoma (Fig. 10, opposite). The optic nerve was divided distal to the mass at the optic foramen, which appeared enlarged, and the right nerve and right half of the chiasma were resected.

On November 21, 1952, visual field of the left eye had improved (Fig. 11*a*, opposite). On December 18, 1953 (Fig. 11*b*, opposite), the visual acuity in the left eye was normal with no additional loss of its remaining field. Examinations in 1954 disclosed no change.

The sequence in this case was one of a progressive chiasmal lesion, beginning prechiasmally on the right and extending to involve the chiasma. Trauma to the head may have produced the haemorrhagic disruption of the nerve and the rapid visual loss.

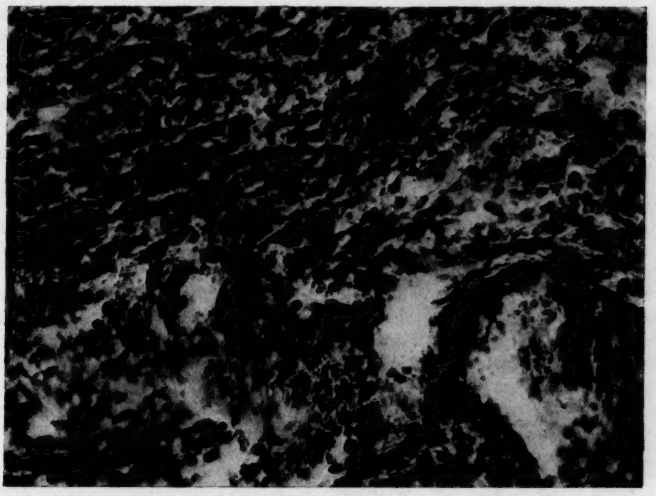


FIG. 10.—Section of pathological lesion in Case 3. Haematoxylin and eosin ×300.

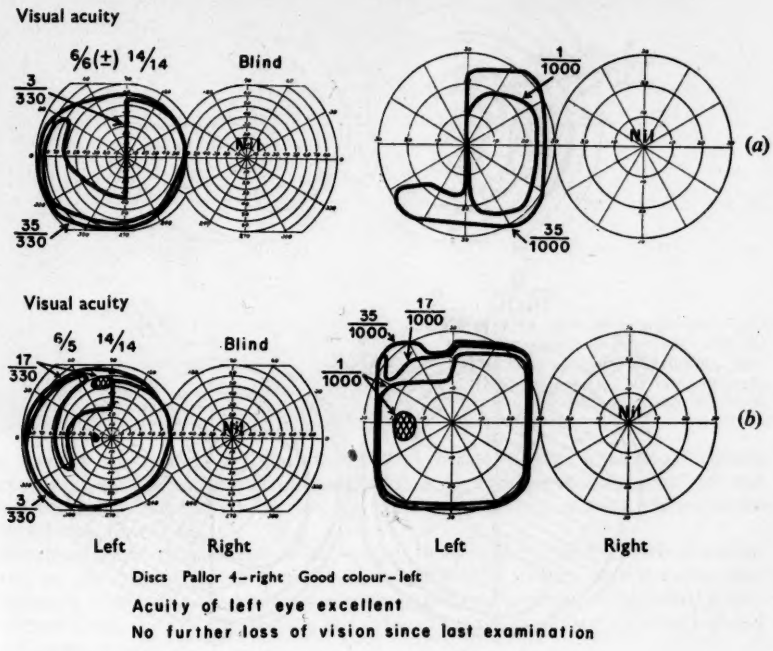


FIG. 11(*a*).—Visual fields in Case 3 plotted on November 21, 1952.
(*b*).—Visual fields in Case 3 plotted approximately 1 year later (December 18, 1953).

Case 4, a woman aged 52, was referred to the clinic with a diagnosis of multiple sclerosis on January 2, 1957. She had noted blurring of vision in the temporal field of the right eye in the summer of 1953, but this impairment of vision had not progressed.

In May, 1956, she had begun to notice blurring of vision in the left eye, and the impairment was progressive until the time of her admission to the clinic. She had been unable to read since November, 1956. Two pneumo-encephalograms, made in the patient's home city, were reported as disclosing nothing abnormal. The content of protein in the cerebrospinal fluid was 107 mg./100 ml.

The results of a general medical examination at the clinic were essentially negative, except for the presence of obesity and hypertension, the blood pressure being 180/130. The patient said she had not had headaches, polyuria, or loss of pubic or axillary hair, or other signs of pituitary dysfunction.

The results of neurological examination were completely negative, except in respect to the visual pathways. On January 3 the visual acuity was 20/70 in the left eye and 20/200 in the right. Grade 2 pallor of the right optic disc and Grade 1 pallor of the left optic disc were noted. The arterioles were narrowed to Grade 1, with minimal sclerosis and focal constriction. The visual fields (Fig. 12) showed bitemporal hemianopia, such as may develop in the presence of a chiasmal lesion. The ophthalmologist could not believe that this was a manifestation of multiple sclerosis.

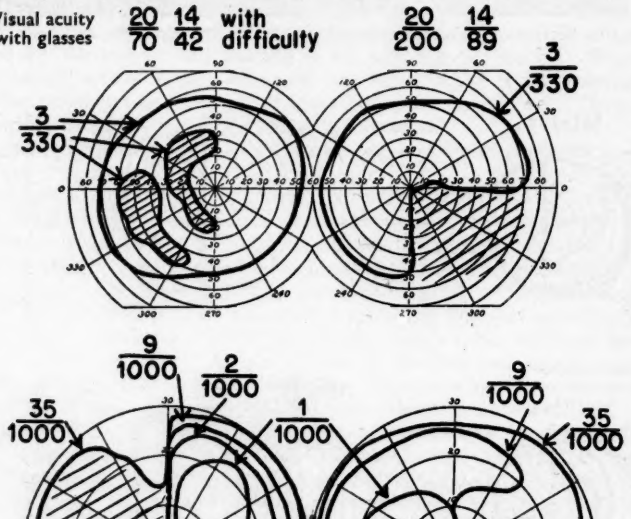
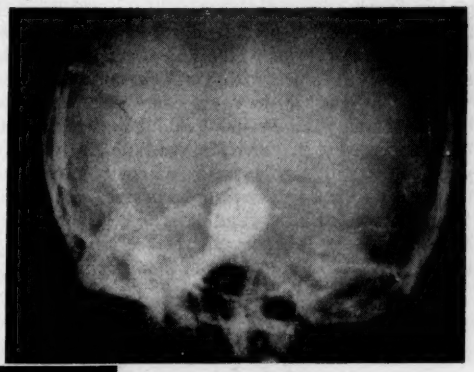


FIG. 12.—Visual fields plotted on January 3, 1957, in Case 4.

On January 7, bilateral carotid angiography was carried out. This demonstrated a large aneurysm of the internal carotid artery beneath the clinoid process (Fig. 13*a*, *b*); there was cross-filling from left to right, but the aneurysm did not fill from the left. It appeared to be bilobate. The interpretation was that the aneurysm might be divided by the chiasma or the optic nerve on the right.



(*a*)

(*b*)

FIG. 13(*a*, *b*).—Antero-posterior and lateral carotid angiograms, with 50 per cent. solution of sodium diacetrizoate (hypaque sodium), demonstrating bilobed aneurysm of right carotid artery.

On January 12, through a right transfrontal craniotomy, an infraclinoid carotid aneurysm was exposed and trapped intra-cranially. It was seen that the aneurysm had elevated the optic chiasma and the right optic nerve, which had grooved the aneurysm into its bilobate form.

Ophthalmodynametric studies done on January 16 showed the diastolic retinal arterial pressure on the right to be 35 mm. Hg and on the left, 52 mm. Hg; 2 weeks after the operation there did not seem to be any noticeable improvement in the visual acuity or the visual fields, even though the size and tenseness of the aneurysm had been reduced by the surgical procedure.

Case 5, a woman aged 56, came to the clinic on June 21, 1956, complaining primarily of recent loss of vision. Her vision apparently had been good until the afternoon of June 17 when she had noticed blurring of vision of both eyes for distant and near objects. She had

been able to read the newspaper that night, but by the next day she could see only the headlines. She had not taken any drugs. There was no history of the use of alcohol or tobacco, or of headaches or other symptoms. Splenectomy had been done in 1953 to relieve secondary thrombocytopenic purpura. The patient recalled that she had used reading glasses for more and more tasks in the previous years, although she had worn glasses intermittently for about 8 years. Physical examination and laboratory tests gave normal results. Roentgenograms of the head disclosed a normal-looking sella turcica and no evidence of erosion of the clinoid processes. The results of the neurological examination, except in respect to vision, were normal. The patient was alert and responded well.

With the right eye she could count fingers at 3 feet, but with difficulty; in the left eye the visual acuity was 20/400. Pupillary reflexes, extra-ocular movements, and peripheral fields appeared to be normal. On June 22 (Fig. 14) the optic discs were found to be of good colour with a rather extensive scotomatous bitemporal lesion due to involvement of the optic chiasma and both optic nerves. The sudden onset and gradual progression in 5 days suggested an expanding lesion, possibly haemorrhage into a pituitary adenoma, an aneurysm, or other prechiasmal lesion. Optic neuritis was considered, but the progressive visual loss seemed to be against this.

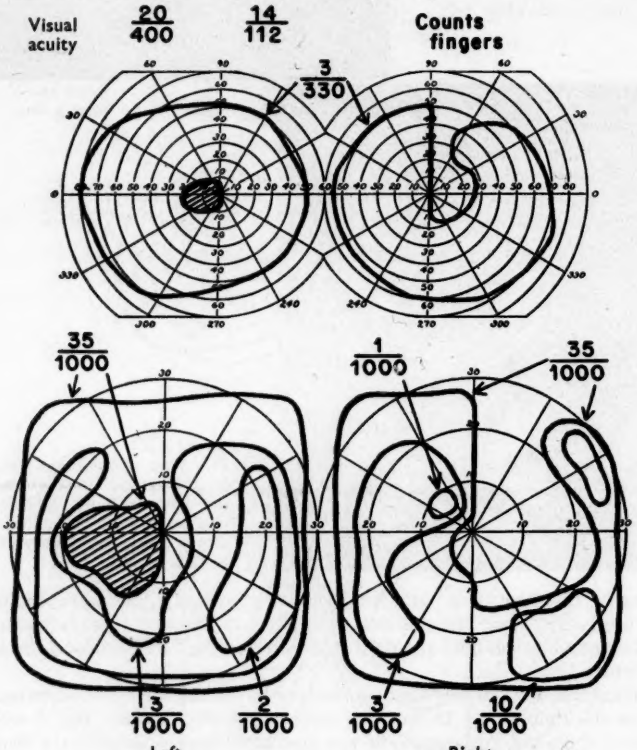


FIG. 14.—Visual fields plotted on June 22, 1956, in Case 5.

The patient was admitted to hospital, and the next morning said she could not see as well as on the night before. Bilateral carotid angiography on June 23 did not reveal any

displacement of the carotid siphon. Because of progressive visual loss, emergency craniotomy seemed indicated. Just before lapsing into unconsciousness for the craniotomy, the patient said she was blind.

A right transfrontal craniotomy revealed no evidence of tumour apart from a mild arachnoiditis about the chiasma and optic nerves. The ophthalmic arteries seemed to be more prominent than usual, and appeared to bow the optic nerves upward. There was no evidence of an aneurysm. Six hours after craniotomy, the patient reported that she could see light with each eye. The pupils were active and equal, and the next day the patient said she could see her own hand as a shadow.

On July 3 (Fig. 15), the visual fields demonstrated a remarkable recovery from the complete blindness of 10 days before. Defects were of the chiasmal type, and at this time the patient could have been recovering from chiasmal neuritis.

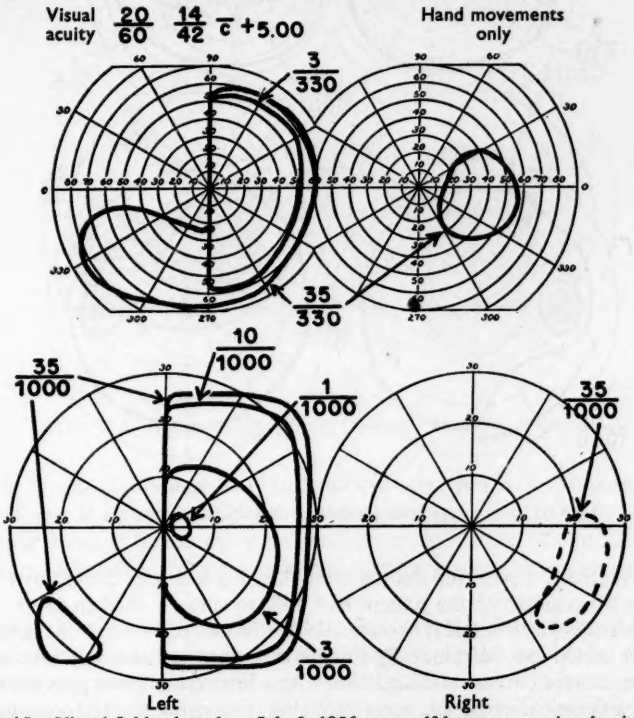


FIG. 15.—Visual fields plotted on July 3, 1956, on twelfth post-operative day in Case 5.

By July 26 the ocular fundi were normal, and the visual fields had almost returned to normal (Fig. 16, overleaf). There was 20/20 vision in both eyes.

The patient did well until about 3½ months later, when vision in the left eye began to fail again. Vision in that eye failed progressively until she presented herself again for examination on November 17, 1956. It was found that the right eye was again blind. Nicotinic acid was prescribed, and the patient again recovered vision gradually. Central scotomata were present in both fields of vision on December 15, 1956 (Fig. 17, overleaf).

On December 22, 1956, it was found that vision in the left eye had continued to improve, though that in the right eye was very poor. On January 5, 1957 (Fig. 18, overleaf),

Grade 1 pallor of both optic discs was seen. The visual field of the left eye continued to improve; the right remained about the same or possibly somewhat worse.

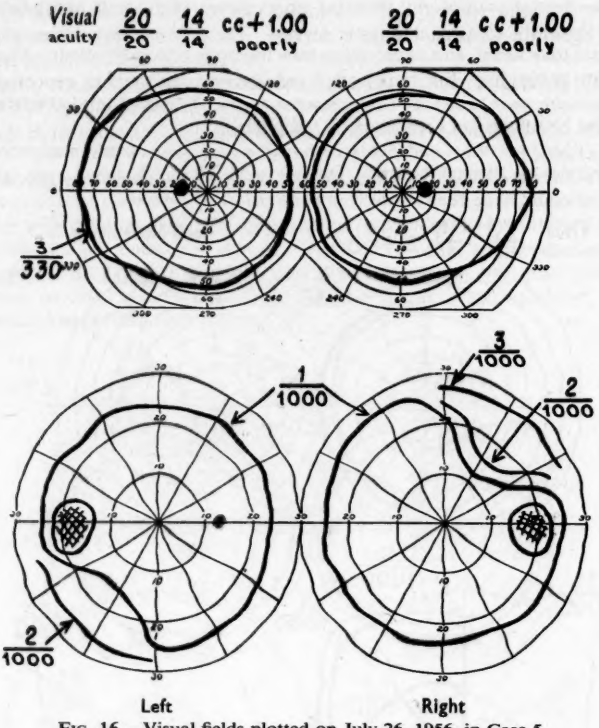


FIG. 16.—Visual fields plotted on July 26, 1956, in Case 5.

It was eventually discovered that chiasmal neuritis had been caused by chemicals containing benzene which the patient had used to spray a chicken coop.

It was felt that early surgical exploration was indicated because of the rapid and progressive visual loss sustained by this patient when no obvious cause could be found after careful clinical investigation. At a later date it was possible to determine that a chemical agent was most probably responsible for the condition.

Summary and Conclusions

An analysis of some of the clinical entities that produce acute visual failure illustrates the need for prompt neurosurgical intervention to prevent irreparable damage to the visual system. The need for a complete and accurate medical, neurological, and ophthalmological investigation in any of these conditions is apparent, and every effort should be made to arrive at the correct diagnosis early, so that definitive treatment can be instituted promptly. A

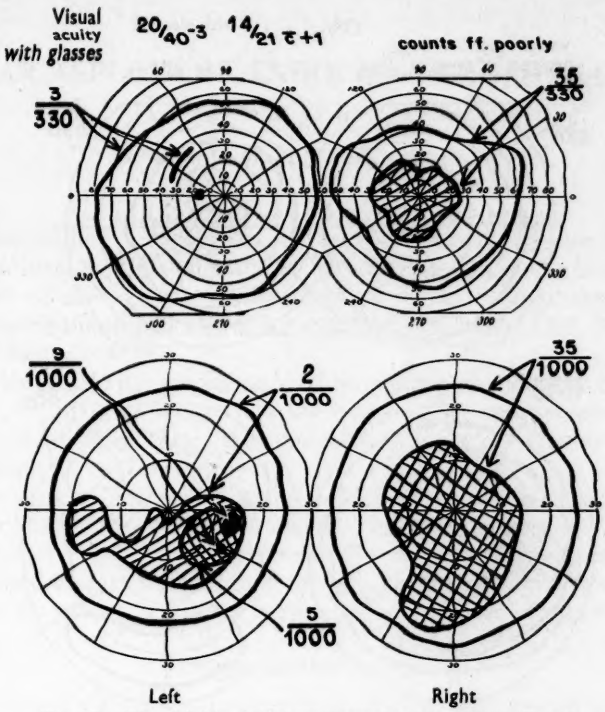


FIG. 17.—Visual fields plotted on December 15, 1956, in Case 5.

diagnosis of retrobulbar neuritis or multiple sclerosis is not uncommon in a hasty evaluation of rapid decrease in visual acuity. This mistake may cause irreparable damage to the optic nerves before the correct diagnosis is made, and surgical treatment may prove discouraging because it is carried out too late.

Five reports of cases are presented to illustrate the difficulties of diagnosis that confront the physician in the management of the problems described. The pitfalls of a hasty diagnosis are obvious, and in many instances definitive treatment can hope only to prevent additional visual loss, since the type of pathological process present of necessity prevents restoration of vision on the affected side. The fact that occasionally an apparently hopeless situation can be retrieved justifies careful search for lesions which can be treated successfully by surgical intervention.

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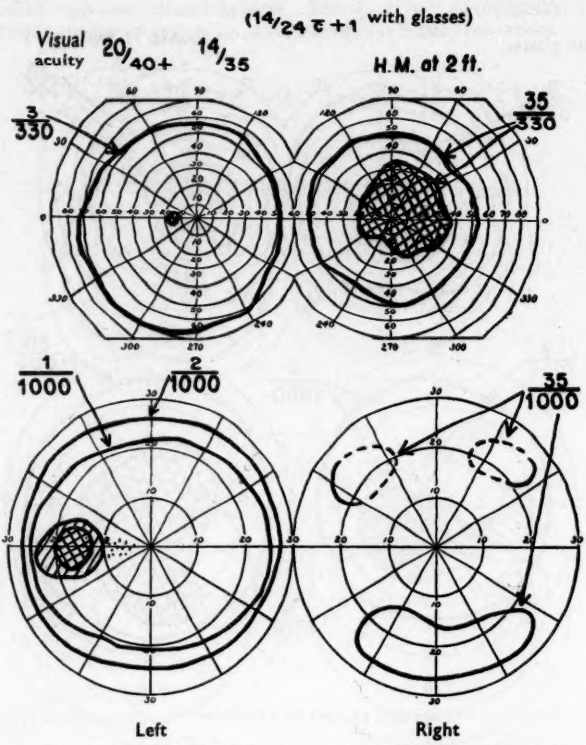

FIG. 18.—Visual fields plotted on January 5, 1957, in Case 5.

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OCULAR ASPECTS OF ENDOCRINE EXOPHTHALMOS*

BY

G. I. SCOTT

Edinburgh

UNDER the heading "endocrine exophthalmos" we may consider on the one hand thyrotoxic exophthalmos and on the other the condition which has been designated exophthalmic ophthalmoplegia, malignant exophthalmos, thyrotropic exophthalmos, and also as a hyperophthalmopathic form of Graves's disease (Means, 1945).

As Wybar (1956) has emphasized, the separation of endocrine exophthalmos into the thyrotoxic form and the malignant or oedematous type is a useful clinical division, since "they undoubtedly represent separate phases of the condition".

Can we then, as clinicians, distinguish between the exophthalmos of acute thyrotoxicosis and that which is potentially "malignant" from the ophthalmic point of view? I think, like Mulvany (1944) and other authors, that there is no doubt that we can, and that from the clinical point of view, from the point of view of the patient, it is of the utmost importance that we should try to do so.

Diagnostic Ocular Signs

(*a*) *Of Graves's Disease.*—In acute thyrotoxicosis the most obvious ocular sign is the lid retraction which varies from day to day. The exophthalmos is often at first more apparent than real and when more advanced is due to "spasm of the anterior orbital unstriped musculature acting in the presence of atonic external ocular muscles", the weakness of the muscles being in part due to the thyrotoxic myasthenia and responding to prostigmine in the early stages (Mulvany, 1944). When the exophthalmos is marked, although the levator may be affected it is never involved without marked impairment of convergence. To these signs we must add the ease with which the eye can be compressed into the orbit.

(*b*) *Of Malignant or Oedematous Exophthalmos.*—In malignant or oedematous exophthalmos, on the other hand, lid retraction is never evident without impairment of upward movement of the eye, nor does it exhibit the great variability from day to day that is so characteristic of acute thyrotoxicosis.

As the condition progresses, the oedema first appears, as Mulvany has emphasized, as a glistening of the bulbar conjunctiva over the insertion of the recti muscles, due to interference with venous drainage through the anterior

* Read at the combined meeting of the Society of British Neurological Surgeons and the 36th Annual Meeting of the Irish Ophthalmological Society in Dublin, May 16–18, 1957.

ciliary veins draining into the swollen muscles. Later still, we have chemosis, congestion, and puffiness of the soft tissues around the eye. The oedema of the lids is often the first obvious sign because of the laxity of the soft tissues, but I have never seen it in the absence of impairment of upward movement of the eye.

In contrast to the exophthalmos of acute thyrotoxicosis the eye is not readily compressed into the orbit.

The difference in compressibility in the two types of endocrine exophthalmos can now be more accurately assessed with the aid of the orbitotonometer (Copper, 1948).

These features have all been clearly described by Mulvany and other authors but bear, I think, re-emphasis. There should, therefore, be little difficulty in recognizing, from the ocular signs, that an exophthalmos is oedematous or malignant in nature.

Classification of Malignant or Oedematous Exophthalmos

Mann (1946) proposed a clinical classification based initially on a report of eighteen cases and later amplified by a study of a further nineteen cases (Mann, 1951). These 37 cases fell into three groups:

(*i*) Those with evidence of primary thyroxine deficiency—with associated excess secretion of pituitary thyrotropic hormone.

(*ii*) Those in which there were initial symptoms of hyperthyroidism followed by spontaneous thyroid failure or failure following thyroidectomy, with an associated excess of pituitary thyrotropic hormone.

(*iii*) Those in which excess of pituitary thyrotropic hormone and thyroxine appeared to arise spontaneously.

To these three groups we may add a fourth:

(*iv*) Euthyroid cases, in which there is no evidence of thyroid dysfunction.

We now know that the problem of malignant exophthalmos involves more than simply a balance between the thyroid and the thyrotropic hormone of the pituitary, and it may well be that malignant exophthalmos is due to some still unknown hormone of the pituitary.

Whatever the cause, the clinical grouping of cases remains unaffected. All these patients present a problem requiring the combined judgement of the physician, surgeon, neuro-surgeon, and ophthalmologist.

Unusual Ocular Aspects of Endocrine Exophthalmos

There are two uncommon aspects to which I should like to make special reference. The first is the type of case in which the exophthalmos may remain unilateral over a long period and in which the diagnostic difficulty lies in deciding whether the case is one of orbital tumour or endocrine exophthalmos. The second is the occurrence of visual field defects in cases of malignant exophthalmos, likewise raising a suspicion of orbital tumour.

(1) Unilateral Exophthalmos.—These cases, although relatively uncommon, present a problem in diagnosis and unnecessary exploration of the orbit may be carried out for suspected tumour. This possibility should be borne in mind in all cases of unilateral proptosis exhibiting slight lid retraction or pseudoptosis (from puffiness of the upper lid), impairment of upward movement, a straightforward proptosis—the difficulty in compression being out of proportion to the degree of proptosis (in the early stages), and absence of any evidence of a soft tissue or other shadow on *x*-ray examination of the orbit.

Hermann (1952) reported such a case in a male aged 63 years, with no symptoms suggestive of thyroid dysfunction, proptosis of the left eye, chemosis, and practically complete absence of upward and outward movement.

X-rays of the skull, sinuses, and optic foramina were negative. "The strictly unilateral proptosis without oedema of the lids suggested the presence of an orbital tumour, although the unusual degree of ophthalmoplegia in the presence of only moderate proptosis raised the possibility of exophthalmic opthalmoplegia which had so far remained unilateral."

Orbital decompression did not reveal the presence of a tumour. The orbital fat was unusually firm and tense, and the muscles appeared unduly bulky. Histological examination of a portion of muscle revealed lymphocytic infiltration with surrounding glandular degeneration of the muscle fibres. At the end of the operation a tarsorrhaphy was carried out.

About 11 months later, the right eye started to protrude. The exophthalmometer readings were 23 mm. in the right orbit, and 15 mm. in the left. *X*-ray treatment of the pituitary resulted in practically complete disappearance of the exophthalmos 2 weeks after completion of treatment, although a slight defect of upward movement and diplopia were still present.

The following two cases which have been under my care are also of interest:

Case 1, a woman aged 49, first examined on January 17, 1956, gave a history of watering of the right eye for 5 months, followed 2 months later by diplopia which was most marked in the mornings and chiefly occurred on looking upwards. At the same time, she noticed some swelling around the eye with protrusion of the globe. Examination showed defective upward movement of the right eye, some retraction of the right upper lid and 4 mm. of proptosis.

The visual acuity was normal in both eyes and the fundi were normal. The blood sedimentation rate was 2 mm./hr. The Wassermann reaction was negative and *x* rays of the orbits and skull showed no abnormality. The history revealed no significant degree of thyrotoxicosis apart from the fact that she was intolerant of heat.

By March 20, 1956, she had developed slight ptosis of the right upper lid and there was evident peri-orbital oedema in addition to the exophthalmos, and also slight chemosis. The blood-pressure was normal and the pulse rate 80. The thyroid gland showed moderate palpable enlargement of the right lobe inferiorly, but no bruit could be detected over it. A radio-active iodine test of thyroid function showed a rather low total excretion of 32·2 per cent., but a normal excretion curve of 6·1, the figures indicating an absence of any peripheral hyper-metabolism.

A biopsy of the right lateral rectus muscle showed some collagenous replacement of muscle fibres with slight cellular reaction.

Arrangements were made for her to have unilateral irradiation of the retro-ocular tissues on the right side, excluding if possible any pituitary irradiation, and this was begun May 1, 1956. *X*-ray therapy was confined to the right lateral field, avoiding irradiation of the pituitary, the patient receiving 3,000 r in 3 weeks. (A field of 4 × 3 cm. was used and no part of the pituitary fossa was included).

Fig. 1 was taken on April 21, 1956, before the irradiation of the orbit was started.

FIG. 1.—Case 1 before orbital irradiation.

When this patient was last examined on February 21, 1957, the proptosis of the right eye was only 0·5 mm. more than that of the left, but there was still slight impairment of upward movement. She said, however, that 4 days before being asked to report for further examination the lids of the left eye had become "puffy" (Fig. 2).

FIG. 2.—Case 1 after orbital irradiation.

In this case the ocular signs have remained unilateral for over a year.

Case 2, a man aged 58 years, first examined on June 6, 1952, gave a history of proptosis of the left eye, commencing 6 weeks previously. The visual acuity and fundi were normal. There was some chemosis of the conjunctiva with 3 mm. of proptosis of the left eye. There was no evidence of hyperthyroidism. *X* rays of the skull and orbits were negative. He was treated with 2 gr. thyroid daily, because of evidence of "thyrotropic exophthalmos".

In January, 1953, the condition was unchanged, and the dose of thyroid was increased to 3 gr. daily.

In April, 1953, the proptosis was much worse, having increased by 2–3 mm.; he was admitted to hospital and while in the ward the chemosis became so marked that it overlapped the lower lid.

X-ray therapy of the left orbit was commenced on May 28, 1953, the posterior limit of the field extending to the region of the anterior clinoid processes: surface dose, 3,250 r. This was followed by an increase of the oedema during the first few days, after which there was rapid improvement, the chemosis and the oedema of the lids disappearing completely. On September 29, 1953, the movements of the eye were normal and there was only very slight exophthalmos. By February 27, 1954, no difference could be detected between the two eyes and when he was last seen in February, 1957, the condition was unchanged.

In this patient the condition has remained unilateral for nearly 5 years.

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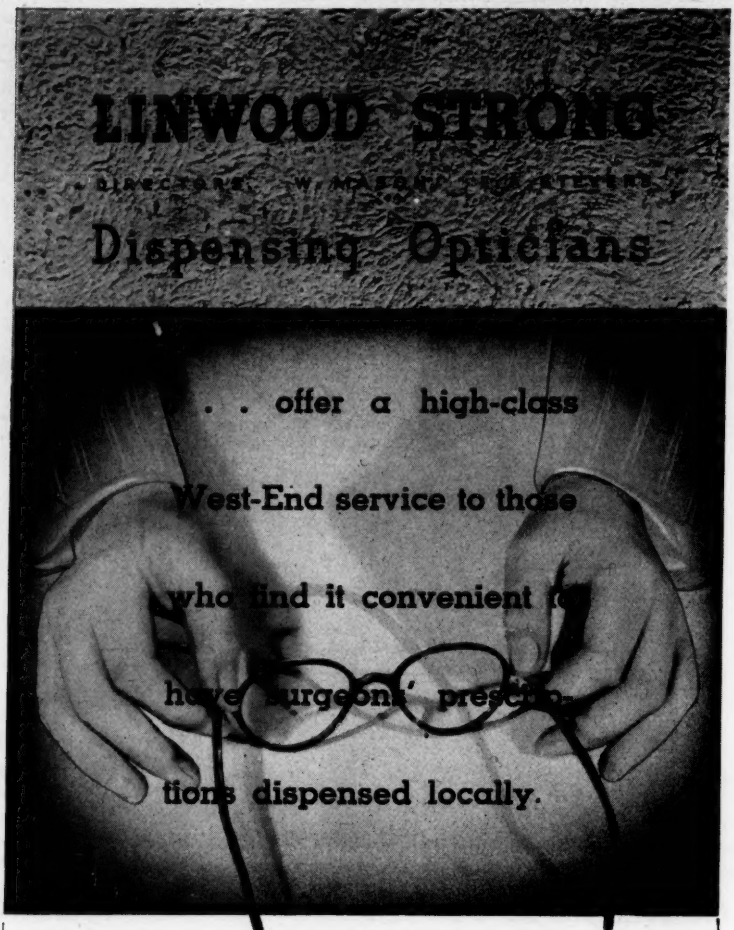

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(2) Field Defects in Malignant Exophthalmos.—Although Naffziger (1933) reported generalized contraction of the fields of vision in two cases of malignant exophthalmos, and loss of the inferior half of the field in another, such cases are very rarely described in the literature.

Igersheimer (1955) reported six cases and Hedges and Scheie (1955) six cases in which defects could be demonstrated in the central fields, indicating involvement of the optic nerve. Of the cases described by Hedges and Scheie (1955), three showed peri-central scotomata, and three showed nerve-fibre bundle defects. All had had hyperthyroidism at some time, although when the field defects occurred, only two were moderately thyrotoxic, two were mildly toxic or in remission, and two were euthyroid.

Naffziger assumed that circulatory impairment in the optic nerves accounted for the defects, since prompt restoration of vision followed when the pressure was relieved by decompression of the orbit. Our experience confirms this, and in this respect the following two cases are of interest:

Case 3, a man aged 54, first seen on March 24, 1954, exhibited definite signs of hyperthyroidism with a moderate degree of exophthalmos. On October 25, 1954, a sub-total thyroidectomy was performed. Early in 1955, signs of ophthalmoplegia became very evident, and the patient was treated with thyroid 2 gr. daily. He developed swelling of the lids, chemosis, and marked interference with all ocular movements. Intravenous ACTH produced some lessening in the degree of exophthalmos. In June, 1955, the proptosis was still increasing and the patient complained of inability to read.

When I first examined this patient on July 30, 1955, the visual acuity in the right eye was 6/9 partly and J. 4, and in the left eye 6/36 partly and J. 20. Examination of the visual fields revealed a central scotoma in both eyes (Fig. 3).

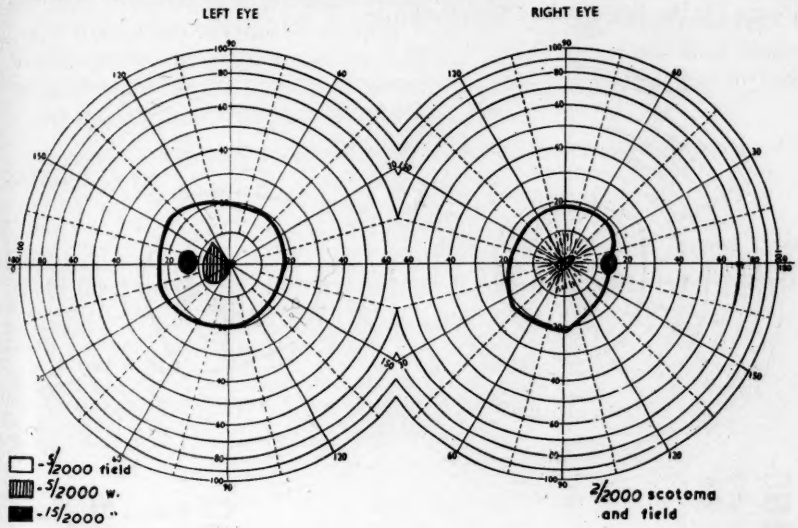

FIG. 3.—Case 3, visual fields on July 30, 1955.

On September 14, 1955, it was apparent that the central scotomata were extending with increasing involvement of macular vision in both eyes (Fig. 4). The visual acuity was 6/9 partly in the right eye and 1/60 in the left eye.

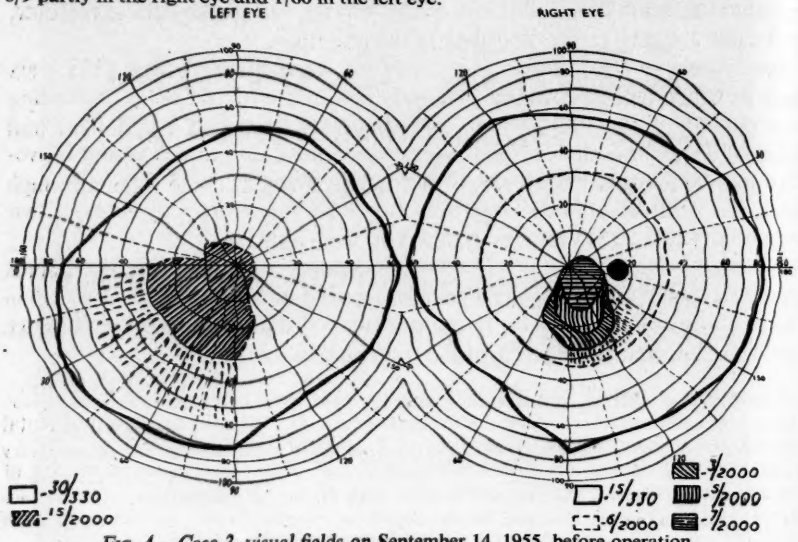

FIG. 4.—*Case 3, visual fields on September 14, 1955,* before operation.

A bilateral orbital decompression was performed on September 22, 1955. On October 5, 13 days after the operation, the charts of the visual fields (Fig. 5) illustrate the dramatic effect of the relief of pressure upon the optic nerves. The visual acuity had improved to 6/9 and J in the right eye and 5/60 in the left.

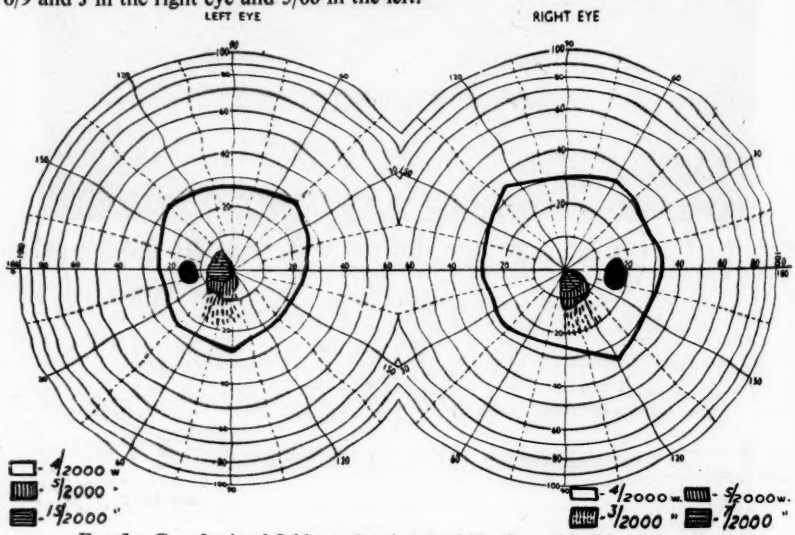

FIG. 5.—Case 3, visual fields on October 5, 1955, after orbital decompression.

Case 4, a woman aged 39, was treated in July, 1955, with thiouracil for early thyrotoxic symptoms, and complained 2 months later of difficulty in focusing the eyes and of diplopia in the mornings. In October, 1955, the visual acuity was 6/9 and 6/6. By December, 1955, she had developed marked proptosis, masked by a tremendously tense oedema of the orbit and eyelids. There was marked limitation of abduction in each eye and of upward movements. The optic discs were normal but the visual acuity had fallen to 2/36 in the right eye and 6/60 in the left. Examination of the visual fields revealed a dense central scotoma in each eye.

Treatment was instituted with intravenous ACTH followed by intramuscular injections of cortisone and ACTH gel. A small dose of radio-active iodine was also given, the patient having been found to be only moderately thyrotoxic. Although the oedema appeared to lessen immediately after the start of treatment, there was no true improvement until April, 1956, when the visual acuity recovered to 6/60 and 6/24. A slow and continuous improvement in vision followed. By July, 1956, the visual acuity was 6/24 and 6/9 and in January, 1957, by which time no field defect could be demonstrated, it was 6/6 and 6/5.

Summary

(1) It is possible from the ocular signs alone to differentiate between the exophthalmos of acute thyrotoxicosis and malignant or oedematous exophthalmos.

(2) The ocular signs in malignant exophthalmos may remain completely unilateral over a number of years.

(3) In rare cases the oedema and infiltration of the orbital tissue in malignant exophthalmos may result in scotomatous defects in the visual fields.

(4) While malignant exophthalmos appears to be a self-limiting disease, irradiation or decompression of the orbit is indicated if the exophthalmos becomes so severe as to endanger visual function.

(5) Irradiation of the orbit seems to be an effective method of treatment, in that it appears to cause a regression of the exophthalmos and thus renders decompression of the orbit unnecessary except in very severe cases or those which do not respond to such treatment.

I am indebted to Professor Riddell and to Dr. R. W. Harrington for permission to quote the findings in Case 4.

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Brit. J. Ophthal. (1958) **42**, 180.

DIAGNOSTIC VALUE OF ORBITAL ANGIOGRAPHY*

BY

H. KRAYENBÜHL

From the Neurochirurgische Universitätsklinik, Kantonsspital, Zürich

THE value of the various methods of *x*-ray examination of the orbit is uncontested for the diagnosis of a space-occupying lesion in the orbit. These methods represent therefore an indispensable diagnostic tool, but nevertheless in many instances the nature of the space-occupying orbital lesion remains obscure. It is the purpose of this paper to discuss the value of orbital angiography as an accessory diagnostic method for such lesions. Curtis (1949), Schurr (1951), and Decker (1955) were amongst the first to describe the angiography of the normal ophthalmic artery and the choroidal plexus of the eye. But angiography of pathological orbital structures is a field which is so far largely unexplored.

In Schurr's opinion the ophthalmic artery is nearly always visible for at least part of its course in lateral carotid angiograms. But in 200 carotid angiograms performed with 35 per cent. contrast medium (Diodrast) we have been able to demonstrate the ophthalmic artery and its branches in only 24 per cent., i.e. in only about one-fourth of our cases. However for the last 2 years we have used the 60 per cent. contrast medium Urografin-Schering, with the result that the ophthalmic artery could be demonstrated in 98 per cent. of 200 carotid angiograms. In 11 per cent. of the cases this artery was visible for only 1 to 2 cm., but in 87 per cent. all the branches up to the frontal branches were demonstrated. In 74 per cent. the choroidal plexus of the eyeball was also seen as a thin crescent in the outer two-thirds of the orbit.

The normal course of the ophthalmic artery is to be seen in the diagrammatic interpretation of Fig. 1 (opposite) and in the arteriograms of Fig. 2 (*a*, *b*, opposite).

Its course is as a rule fairly constant. It is seen to arise from the internal carotid as it emerges from the cavernous sinus close to the anterior clinoid process. According to Schurr, the artery pursues a slightly tortuous course largely because of the freedom of movement which must be allowed to its branches in order to permit rotation of the eyeball. It is unlikely, therefore, that displacements by space-occupying lesions within the orbit would be easy to detect. The recognition of the different branches of the ophthalmic artery and of the choroidal plexus is not easy, especially in distinguishing them from the outlines of the bone which overlies them. In our experience the stereoscopic views of the angiograms were of great help in the demonstration of these vessels.

* Read at the combined meeting of the Society of British Neurological Surgeons and the 36th Annual Meeting of the Irish Ophthalmological Society in Dublin, May 16–18, 1957.

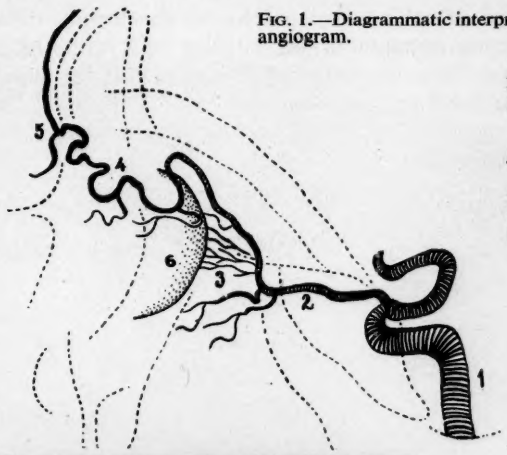


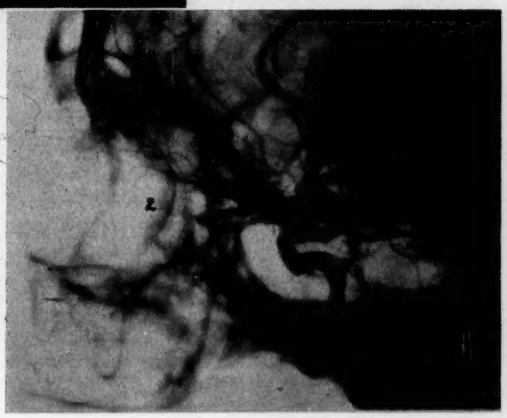
FIG. 1.—Diagrammatic interpretation of a normal orbital angiogram.

1. Internal carotid artery.
2. Ophthalmic artery.
3. Anterior and posterior ciliary arteries and central artery.
4. Internal palpebral branches.
5. Frontal and dorsal nasal artery.
6. Choroidal plexus.

(*a*)

FIG. 2 (*a*, *b*).—Lateral arteriogram showing tortuous course of ophthalmic artery (1) and choroidal plexus (2).

In Fig. 2 (*b*) a loop of the artery is to be seen.



(*b*)

As a second method of orbital angiography we developed orbital venography by percutaneous injection of the angular vein with the contrast medium. The method has some technical difficulties. If the angular vein is very thin it must be punctured with the head in a downward position and with compression of the facial and frontal veins. The course of the orbital veins is to be seen in the phlebogram Fig. 3 (*a*, *b*).

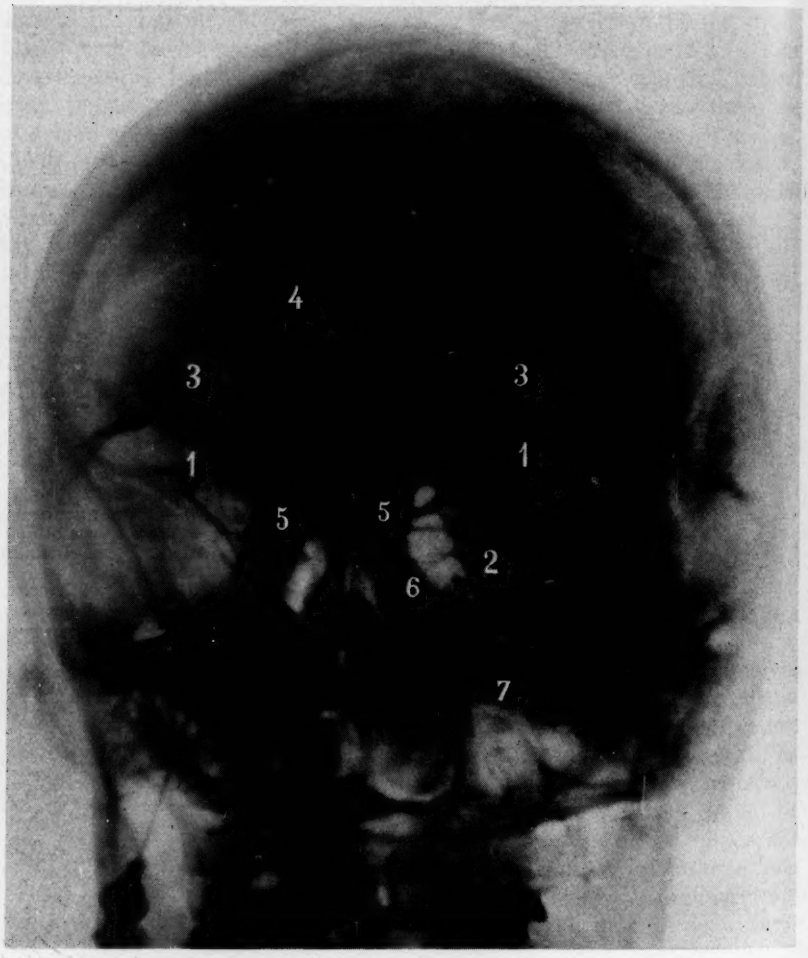


FIG. 3 (*a*, *b*).—Antero-posterior and lateral phlebogram showing the superior (1) and inferior (2) ophthalmic veins, and the supra-orbital (3), frontal (4), angular (5), nasal (6) and anterior facial (7) veins.

FIG. 3 (*b*).

1.	Intra-orbital Tumours	22
2.	Peri-orbital Tumours	36
3.	Vascular Diseases:	
	(*a*) Intracranial saccular aneurysms	7
	(*b*) Intracranial arteriovenous aneurysms	2
	(*c*) Traumatic fistulous carotid-cavernous aneurysms	21
	(*d*) Orbital varix	4
4.	Inflammatory Processes:	
	(*a*) Acute	3
	(*b*) Chronic	2
	(*c*) Thrombosis of the cavernous sinus	3
5.	Graves's Disease	5
6.	Miscellaneous	12

Our case material, which has been analysed by Yaşargil (1957), consists of 117 cases of unilateral proptosis. The underlying pathological disease in each case is shown in the Table.

From this material were taken the following nine cases of unilateral exophthalmos in which the cause was detected by orbital angiography.

These appear to be amongst the first such cases to be reported in the angiographic literature.

FIG. 4.—Lateral angiogram demonstrating a large diffusely-stained, retrobulbar melanoma in Case 1.

Case Reports

Case 1, a 46-year-old male, had suffered from slowly progressive left-sided proptosis with gradual failure of vision for 8 years. There was a huge left orbital tumour and proptosis, left papilloedema, and visual loss (1/60). *X* ray of the left orbit showed some increase in density of the orbital contents. Left carotid angiography revealed a marked forward and upward displacement of the choroidal plexus of the left eyeball and a huge retrobulbar diffusely-stained tumour (Fig. 4).

The ophthalmic artery was very thin and not displaced, but the internal maxillary artery was considerably enlarged.

The tumour, a huge melanoma, was removed at operation.

The patient died 6 weeks later from general metastases.

Case 2, a 2-year-old boy, was noted to be developing a left-sided parenchymatous keratitis and a gradually increasing left-sided proptosis after an alleged injury of the left eyeball with a pencil 8 weeks before. Numerous ophthalmologists were consulted, and the diagnosis was made of primary or secondary glaucoma and of an abscess of the lens. 9 months after the onset of the illness, on October 5, 1956, the left eyeball was finally removed and a prosthesis was implanted. But there was a progressive protrusion of the prosthesis combined with staphylococcal infection, and the patient was admitted to the neurosurgical clinic for orbital angiography. A retrobulbar tumour or abscess was diagnosed.

The left carotid angiogram (Fig. 5) demonstrated a huge tumour with numerous, very fine pathological vessels in the left orbit and no displacement of the ophthalmic artery.

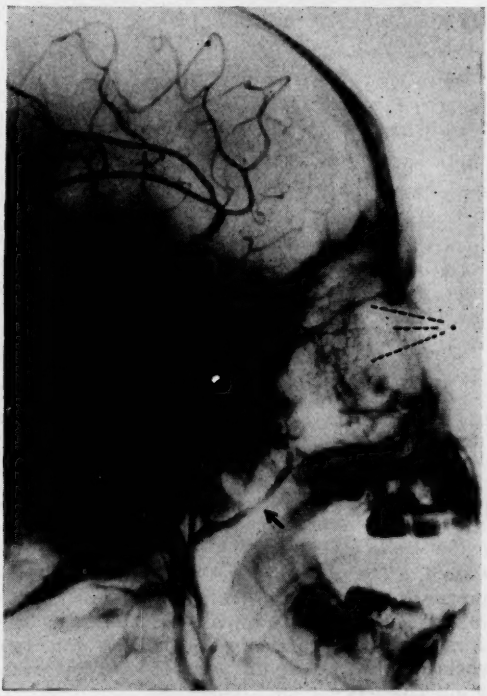


FIG. 5.—Lateral angiogram showing a large orbital retinoblastoma with fine pathological vessels and enlarged internal maxillary artery (arrow) in Case 2.

On December 21, 1956, the tumour was removed totally by Prof. M. Amsler, and proved to be a retinoblastoma. The post-operative course was uneventful and post-operative *x*-ray treatment was instituted.

Case 3, a 52-year-old woman, had suffered from diplopia and slight protrusion of the left eye for a few months. There was proptosis of 4 mm. and downward displacement of the left eye, as well as some impairment of adduction and abduction, and hypaesthesia in the left first trigeminal division. *X* rays of the skull and orbit were normal.

The left carotid angiogram (Fig. 6, overleaf) showed a normally placed left ophthalmic artery which gave use to abnormal branches surrounding a tumour the size of a cherry behind the eyeball. The forward displacement of the choroidal plexus is well seen. As the patient's general condition was not good, no operation was performed. The patient died 6 months later from carcinoma of the lung, of which the retrobulbar tumour was a metastasis.

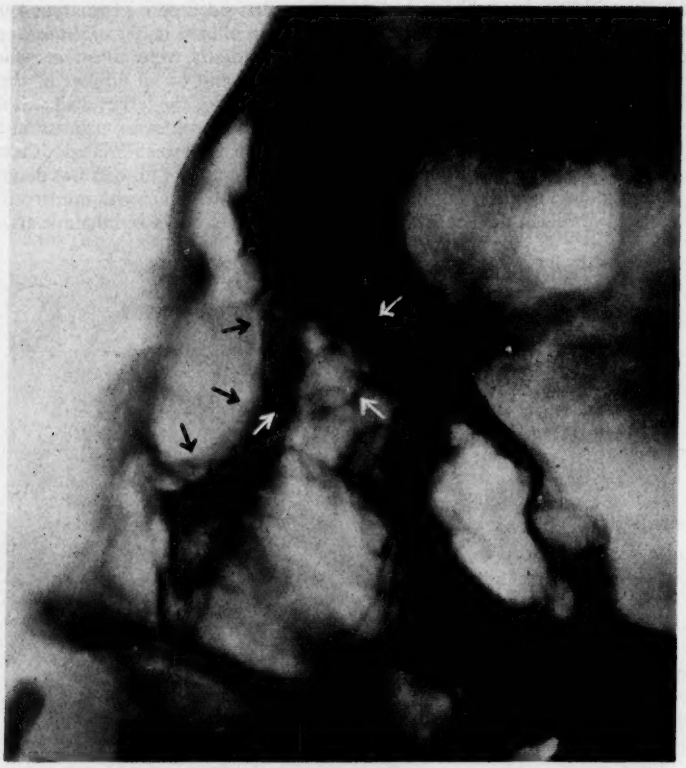


FIG. 6.—Lateral angiogram showing a retrobulbar encapsulated tumour and displaced choroidal plexus in Case 3.

Case 4, a 6-year-old girl, had been suffering from right-sided headache, diplopia, and progressive protrusion of the right eye for 4 weeks. There was considerable proptosis of the right eye with downward displacement and impairment of all ocular movements. *X* rays of the skull, including tomography of the orbit, were normal. The right carotid angiogram (Fig. 7, opposite) showed a retrobulbar tumour, the size of a cherry, without displacement of the ophthalmic artery.

In the venous phase of a right external carotid angiogram, the facial, angular, nasofrontal, and superior ophthalmic veins were demonstrated as well as the downward displaced choroidal plexus (Fig. 8, opposite). At operation (orbitomy) the tumour was found to be well encapsulated and was radically removed. Histological examination showed it to be a retinoblastoma. A post-operative course of *x*-ray treatment was given.

Case 5, a 44-year-old man, had suffered from a gradually increasing protrusion of the right eye for 2 years. There was right-sided exophthalmos of 6 mm., downward and inward displacement of the right eye, and complete paralysis of the right sixth nerve. The right carotid angiogram (Fig. 9, opposite) showed an avascular tumour with fine capsular blood vessels between the thinned and elevated orbital roof and the ophthalmic artery. The choroidal plexus was well shown and downwards displaced. A clinical diagnosis of a lipoma or dermoid cyst was made. The patient refused a surgical exploration.

FIG. 7.—Lateral angiogram showing a retrobulbar retinoblastoma in Case 4.

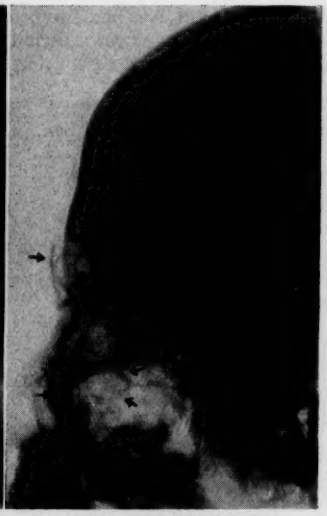


FIG. 8.—Orbital phlebogram showing the downward displaced choroidal plexus in Case 4.

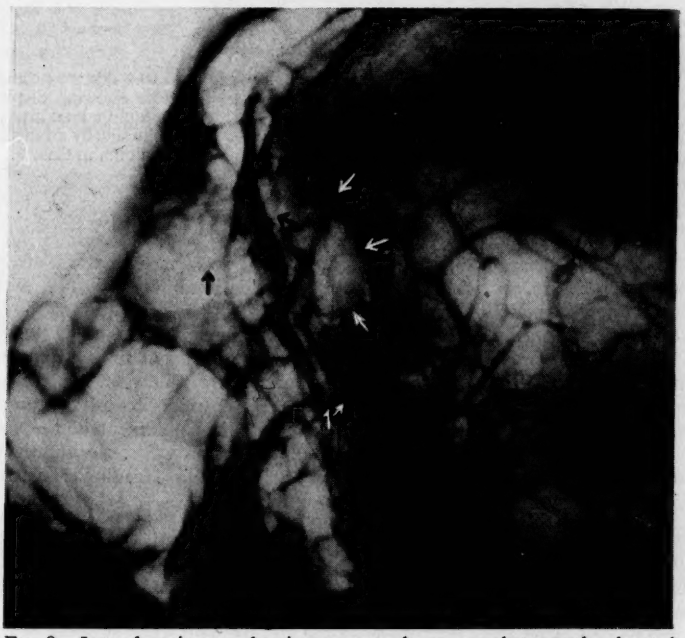


FIG. 9.—Lateral angiogram showing an avascular tumour between the elevated orbital roof and ophthalmic artery in Case 5.

Case 6, a 3-year-old girl, was noted to be developing a swelling of the upper eyelid and a protrusion of the right eye over a period of one year. There was a considerable right exophthalmos of 7 mm., and right primary optic atrophy with normal vision. Radiography of the orbit showed an enlargement of the right optic foramen. In the antero-posterior view of the right carotid angiogram (Fig. 10) a medial displacement of the right ophthalmic artery at its origin was detected. At operation a glioma of the optic nerve was found and removed.

FIG. 10.—Antero-posterior angiogram showing displacement of the ophthalmic artery to the opposite side by an intra-orbital optic glioma in Case 6.

Case 7, a 54-year-old man, *had sustained a motor-cycle accident* with a basal fracture of the skull 4 years before, and had been suffering from tension in the right eye, swelling of *the right lower eyelid, and diplopia for 6 months.*

There was right proptosis of 7 mm., upward displacement of the right eyeball, and *slight right sixth nerve* weakness. At the base of the orbit a tumour was palpated and tomography of the orbit revealed destruction of the floor of the orbit. In the capillary and venous phase of the right carotid angiogram (Fig. 11, opposite) a huge tumour was seen with peripheral, capsular vessels and an upward displacement of the choroidal plexus. The diagnosis of a peri-orbital and maxillary tumour was made, and the tumour was radically removed from the maxillary sinus by Prof. Rüedi. *Histological examination* showed it to be a neurinoma of the right infra-orbital nerve.

Case 8, a 59-year-old man, had sustained an accident to the right temple with injury of the right eye and fracture of the right orbit one year ago. Immediately after the accident he was blind in the right eye, and there was a complete right-sided ophthalmoplegia; 4 months later the right eye was removed because of progressive panophthalmitis. At the same time he developed some protrusion of the left eye with some swelling of the veins of the eyelids. After 6 months an intracranial bruit and a slight left-sided exophthalmos

FIG. 11.—Lateral angiogram showing a large periorbital neurinoma of the infra-orbital nerve surrounded by the terminal branches of the internal maxillary artery (1) in Case 7.

with vascular engorgement of the eyeball were detected, and a traumatic fistulous carotid-cavernous aneurysm was diagnosed. The angular and carotid phlebograms (Fig. 12 *a, b*) showed an enormous dilatation of the angular and superior ophthalmic veins up to the cavernous sinus.

(*a*) (*b*)

FIG. 12.—Angular and carotid phlebograms showing very marked dilatation of the angular and superior ophthalmic veins in a case of traumatic fistulous carotid-cavernous aneurysm (Case 8).

Case 9, a 35-year-old woman, was developing a slight swelling of the left eye with impairment of vision on bending the head forward. There was a very slight left proptosis, and a slight increase of the exophthalmos on forward inclination of the head. *X* rays of the skull and orbit, and right carotid angiogram were normal. Percutaneous venography of the left angular vein (Fig. 13) demonstrated a cherry-like enlargement of the left superior orbital vein, and an orbital varix was diagnosed.

FIG. 13.—Antero-posterior angular phlebogram showing an intra-orbital varix of the superior ophthalmic vein.

Definite conclusions cannot yet be drawn from this relatively small series regarding the diagnostic value of orbital angiography. But as Schurr has pointed out this method deserves the attention of ophthalmologists, radiologists, and neurosurgeons. With further experience the method may be used to demonstrate and to distinguish both orbital and peri-orbital neoplasms and vascular abnormalities. This series seems to show that the value of orbital angiography lies most likely in the demonstration of the pathological vascularization of the space-occupying lesion and not of the displacement of the ophthalmic artery and its branches.

Summary

The angiographic appearances of the ophthalmic artery and the choroidal plexus of the eye and also the orbital veins are described by the method of orbital angiography. The value of orbital angiography is discussed with regard to a small series of orbital space-occupying lesions.

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BOOK REVIEW

Traquair's Clinical Perimetry. By G. I. Scott. 7th ed., 1957. Pp. 333, 274 figs, bibl. Kimpton, London. (60*s.*).

It is now 30 years since the appearance of the first edition of "An Introduction to Clinical Perimetry" by H. M. Traquair. Its message to ophthalmologists was the importance of quantitative perimetry and the interpretation of results produced by a technique which, in modern times, might be called reconnaissance in depth. As the years passed Traquair added more and more to his store of personal observations and his book in the end contained a vast amount of information. Prof. G. I. Scott has just edited and published the seventh edition, which he has renamed "Traquair's Clinical Perimetry". He has taken, as his main purpose, a simplification of presentation, in order that the basic principles of perimetry may never be obscured. It is the reviewer's opinion that there was need for simplification, remembering weary hours struggling to understand the intricacies of complicated fields of vision. The nature of perimetry itself contributes to the difficulties encountered by the student, because it is an art which cannot be learnt from a textbook. In this aspect of ophthalmology the teacher comes into his own and a good hour of practical instruction is worth nights of solid reading. Just as it is difficult to learn the art of perimetry book-wise, it is almost impossible to understand the full significance of pathological fields of vision until some practical experience has been gained with the Bjerrum screen.

In this edition, which follows a similar pattern to its predecessors but which flows more smoothly and is beautifully illustrated, there is a fuller discussion of the applied anatomy of the visual pathway. There are alterations in the sections on optic neuropathy and in the chapter on affections of the anterior part of the optic radiation. It is interesting to find that Scott admits that the use of projection apparatus has some advantages although it seems unlikely to replace entirely the classical methods of examination, and at last, we find a picture of a perimeter which has a self-recording apparatus attached to it. It may be true that all great perimetrists have advocated the use of simple non-mechanical instruments, but we are not all great perimetrists or ever likely to be so, and mechanical instruments are well suited to the routine clinical work of an out-patient department, especially when there is any kind of pressure for time.

In the last 30 years the position of perimetry as a means of diagnosis in neurology has changed. It has now to share its place with air encephalography and angiography in the preliminary investigations and, whereas these new techniques have been revolutionary in their methods and in their precision, perimetry has advanced in refinement only through collation of fields of vision with autopsy findings. This does not in any way underestimate the importance of perimetry; indeed, a defect in the field of vision is often the first step towards the elucidation of a disease process in the central nervous system, and this being so, one would welcome, from a purely practical point of view, a chapter on the factors which induce an ophthalmologist to undertake detailed exploration of the visual fields. Anyone who has seen large eye hospitals at work realizes that there must be some guiding principles which will help the consultant to decide which of their patients need this time-consuming investigation. This would be a difficult chapter to write but, unless there is some answer to such a challenge, it might be supposed that the decision to employ perimetry was guided by guess or by God, an unbearable conclusion to have to draw.

Traquair was probably most fortunate in this particularly subjective type of testing in having as his patients the Scots in their capital city. They see things in black and white with hard margins, and if they cannot, have no hesitation in saying "No", without the soothing cadence of a diphthong. Their lack of compromise makes them good perimetric subjects and from brief encounters with Traquair at the meetings of the Scottish Ophthalmological Club during the war, the reviewer would suspect that he gained much

satisfaction from blasting their answers upon the sands of time in deep and unequivocal furrows. The new editor has earned our gratitude by tidying and bringing up to date this monument of industry and careful observation, and has made sure that the seventh edition is the best available guide for the understanding of the visual field in health and in disease.

NOTES

Oxford Ophthalmological Congress

43rd Annual Meeting, 1958

Master .....Sir Tudor Thomas
Deputy Master .....C. H. Bamford
Editorial Secretary .....L. P. Jameson Evans
Hon. Secretary and Treasurer.....Ian C. Fraser

The Oxford Ophthalmological Congress will again assemble at Balliol College, Broad Street, Oxford, on July 6 and meetings will be held on July 7, 8, and 9, 1958, in the Lecture Hall of the School of Physiology, South Parks Road, Oxford.

The Doyne Memorial Lecture will be delivered by Mr. O. Gayer Morgan (*London*) and will be entitled "The Early Clinical Diagnosis of Glaucoma".

There will be discussions on "The Influence of Vascular Changes in Progressive Failure of Vision", and "The Aetiology and Treatment of Uveitis"; any member wishing to contribute to these discussions should send his name to the hon. secretary.

It is proposed to repeat the successful "Any Questions" session and members are invited to send in queries for debate before the meeting.

Ophthalmologists not already members of the Congress who wish to attend the meetings should communicate with the hon. secretary. There is no entrance fee or annual subscription, but a fee of three guineas is payable on each occasion of attendance at a Congress.

OBITUARY

William Gordon Matthew Byers

Every British ophthalmologist will regret the death of Gordon Byers on August 2, 1957. He was born in Ontario in 1872 and graduated in medicine from McGill University in 1894, taking a post-graduate course thereafter in Edinburgh and serving for 18 months as a house-surgeon at Moorfields. Following this he studied for a year in Berlin, Paris, Vienna, and Utrecht, and on his return to Canada from Europe, joined the staff of the Royal Victoria Hospital, Montreal, in 1898. He became senior ophthalmologist in 1921 and professor of ophthalmology at McGill in 1923. He retired from the hospital in 1935, from the university in 1937, and from private practice in 1946.

Byers probably did more than any other to establish Canadian ophthalmology on a sound footing, and the debt that Canadian ophthalmology owes to him is immense. In 1920 he was essentially responsible for founding the Montreal Ophthalmological Society and became its first president, and in 1937 he was largely responsible for founding the Canadian Ophthalmological Society and was again its first president. In 1934 he was president of the American Ophthalmological Society and he was also a member of the Ophthalmological Society of the United Kingdom from 1897. Exact and methodical in his work, concise and punctilious in his writings, beneath a certain stiffness of manner, he was essentially one of the most kind and generous of men. Apart from ophthalmology, he had many hobbies, all of which he embraced with enthusiasm—gardening, farming, bird-watching, and sailing. He is survived by his wife, two sons, and two daughters; one son was killed while serving with the Canadian Royal Air Force in the second world war.